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VOL. II.—14TH YEAR. SYDNEY: SATURDAY, AUGUST 20, 1927. No. 8.


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## ACUTE DILATATION OF THE STOMACH DURING LABOUR AND THE PUERPERIUM.

By BRIAN H. SWIFT, M.C., B.A., M.B., B.C. (Cantab.), F.R.C.S. (Edin.),
*Obstetrician, Queen's Maternity Home, Adelaide; Tutor in Obstetrics, Adelaide University.*

THE condition of acute dilatation of the stomach is a moderately common sequel in patients who have had abdominal operations and the first description which I can find was given by Hilton Fagge[1] in 1872 when he described several cases. However, the condition is very rare in connexion with labour and the puerperium and few cases have been reported. I have consulted various English textbooks on obstetrics and the subject has not been mentioned either as a pathological condition of labour or of the puerperium or as a cause of death after labour. Whitridge Williams[2] in his latest book writes: "The condition is most serious . . . and it should be recognized as one of the causes of sudden death following delivery."

The German textbooks such as those by Stoeckel, Menge-Opitz, Döderlein, Bumm, do not mention the condition, nor does the largest Austrian book by Halban and Seitz lay any stress on it. Amongst the authors of the American textbooks, De Lee[3] wrote that vomiting during labour was common and he gave a list of the causes, but this list did not include acute dilatation of the stomach as a sequel of the vomiting. Later he mentioned under the heading of "Sundry Complications of the Puerperium"[4] that tympany might occur after labour, but that it seldom became obstinate or very pronounced and he did not worry about it, unless the patient's general condition was affected. He mentioned acute dilatation only in connexion with ileus after Cæsarean section and stated that "acute dilatation may be the cause of the ileus." Here, however, he was dealing with a postoperative form of acute dilatation.

Having regard to the slight reference to the condition in the large textbooks, it must be admitted that either the condition is one of very great rarity or that it has escaped notice or, as Audebert[5] stated, "a large number of dilatations are unrecognized, being confused with post-chloroform vomiting or some peritoneal syndrome."

Numerous articles have been written on the subject of acute dilatation of the postoperative variety, but in connexion with labour I can find few references. Audebert[6] in 1912 gave a full account of cases published in France and he was able to collect only ten. Several of these also were postoperative and so should not be counted. I will, however, refer to this later.

Thomson[7] in 1902 published a case, but I have been unable to find an earlier publication. Berkeley and Bonney[8] in their book on the "Difficulties and Emergencies of Obstetric Practice," quote a case. In 1907[9] Kuschnir reported a case and in 1911 Hellendall[10] published an article in which he had collected one hundred and forty cases, in which fifty-four patients had had no operation. He came to the conclusion that the cause was always due to some form of sepsis. However, no details were given of the individual patients nor was any mention made of the patients being in labour or the puerperium.

In 1913 Davis[11] reported a case and in South America Ramos [12] published an article in which he had collected seventeen cases, but a number of the patients in this series had been subjected to Cæsarean section. In the Dutch literature I have found a case quoted by Van der Perk.[13]

Later on in the paper I will collect and tabulate these cases, omitting those in which the patients have had operations such as Cæsarean section, as I consider them to be a postoperative variety. As the condition is so rare and so little has been noted in it, I consider that the case which occurred in my own practice to be worth reporting in detail.

### Report of Case.

Mrs. L., aged twenty-eight years, was a *primipara*. She was a strong, healthy woman with a history of no previous illnesses, had had an uneventful pregnancy. Antenatal examination had revealed nothing abnormal. She had arranged to have her baby in her own house and labour commenced on June 17, 1923, at 3 p.m. (This was about the calculated day.)

I saw her at 6.30 p.m., when her pains were not very strong. By palpation the head was found to be engaged and in the left occipito-anterior position. She had feeble pains all that night and during the next day. I saw her at 6 p.m. next day and found that she had not beeen able to pass her urine and the bladder was catheterized. She had been sick and vomited during the day after all food and drink. She had had some trouble with her nurse and it was decided to transfer her to hospital. She was admitted to hospital at 9.30 p.m. on June 18, when she was having pains about every ten minutes. She was given an enema with a good result.

I saw her at 10.30 p.m. and was informed that she had vomited a little greenish fluid since admission. The head appeared to be descending and on vaginal examination the *os* admitted two fingers and cervical canal was obliterated. At 11.30 p.m. on June 18 she was given morphine fifteen milligrammes (one quarter of a grain) and hyoscine 0·6 milligramme (one-hundredth of a grain) by hypodermic injection. She slept for about four hours and at 5 a.m. on June 19 the membranes ruptured, her temperature was 36·7° C. (98° F.) and the pulse rate 88. At 7 a.m. she vomited some brownish fluid. She was having good pains and was dozing between the pains.

I saw her at 10 a.m. and examined her *per vaginam* and found that the *os* still admitted only two fingers and that its edges appeared to be very hard. As she had had strong pains, I decided to give her hot douches to try to soften the cervix.

She vomited again at 11 a.m. when her pulse rate had risen to 112. The vomit was thin and of a light brown colour. At 2.30 p.m. she vomited and again at 3.30, when she vomited about three hundred cubic centimetres (ten ounces) of brown fluid. She was having soda water to drink and was very thirsty and she vomited again two hundred and ten cubic centimetres (seven ounces) of brown fluid at 4 p.m. At 4.15 p.m. her pulse was 124 and her pains were very severe.

I saw her at 4.30 p.m. and the fœtal heart was heard with a Pinard's stethoscope and was 140 and regular. Her temperature was 37·2° C. (99° F.) and her pulse rate 118. I was very much tempted then to dilate the cervix and extract the child. However, I decided to wait and see her later. At 5.30 p.m. she vomited two hundred and forty cubic centimetres (eight ounces) of brown thick fluid; this was quite like a coffee-ground vomit. I saw her again at 7.30 p.m. and repeated the morphine and hyoscine and she settled down. At 9.30 p.m. she was complaining of pain in the back and across the chest and at 9.45 she vomited two hundred and ten cubic centimetres (seven

ounces) of light-brown vomit. The pains were becoming more severe, but she was sleeping between them.

At 2 a.m. on June 20 the pains were more severe and I ordered morphine ten milligrammes (one-sixth of a grain) and hyoscine 0·3 milligramme (one two-hundredth of a grain). At 8 a.m. I saw her and examined her and found the *os* nearly fully dilated, a small edge could be felt all round. The pains were very strong and the head had come well down. At 10.30 the head was on the perineum. I decided to apply forceps to help the head over the perineum. A catheter was passed and two hundred and ten cubic centimetres (seven ounces) of urine were drawn off. The slightest amount of chloroform was given and with a very little force the baby was born. The baby was oligopnœic and a little blue, but I realized that this was due to the morphine and the baby quite recovered in about an hour without much stimulation.

Immediately after the child was extracted and the cord was tied, the nurse who had had her hand on the woman's abdomen, turned to me and said, "Something has happened, she has become suddenly very big." I at once inspected the abdomen and found the whole abdomen was tremendously distended and on percussion was very resonant. The distension was very pronounced in the upper part of the abdomen. However, the abdominal cavity was so tense that it was absolutely impossible to feel the body or fundus of the uterus, but yet there was no rigidity of the muscles. At the time I could not think what had happened. I thought of rupture of the uterus with air entering the abdominal cavity, but her general condition was so good. The uterus was bleeding a little and as I could not feel the fundus, I decided to remove the placenta manually. She was given a little more chloroform and the placenta was easily removed and one stitch put in the torn perineum.

"Pituitrin" one cubic centimetre was given intramuscularly at 11 a.m. and it was repeated again at 11.20 a.m. She had stood the delivery very well, looked in good condition and her pulse was only 100. I decided as she appeared so well that the distension must be due to some sudden dilatation of the stomach and intestines.

She was put back to bed at 11.30 a.m. At 1.30 p.m. she vomited brown fluid, but afterwards managed to take sips of water without vomiting.

At 5 p.m. her condition became much worse. Her temperature was 37·5° C. (99·6° F.) and her pulse rate 140 and she was given 0·5 cubic centimetre of "Pituitrin" every two hours. At 8 p.m. she began to complain of very bad abdominal pain. I decided this was due to her abdominal distension which was, if anything, greater. She was given a high turpentine enema with a good result and passed flatus freely and she passed urine.

On the second day at 2.45 a.m. (June 21), that is about fifteen hours after delivery, she complained of pain in her chest and vomited two hundred and ten cubic centimetres (seven ounces) of thickish brown fluid. Her pulse was 120 and temperature 36·9° C. (98·4° F.). The abdominal distension was no less.

Her temperature continued to drop and she complained of great pain and at 8 a.m. her temperature was 35·1° C. (95·2° F.) and her pulse rate was 120. She was given brandy every hour. Her condition now was very serious; the enormously distended abdomen was giving her pain, the pulse was very rapid and the temperature was subnormal. At 9.30 a.m. she passed a very large motion with much flatus. The vaginal discharge had been normal all this time, so I decided that the uterus, which I could not feel, was most probably involuting normally and that I had to contend with some form of paralytic distension of the stomach corresponding to an acute dilatation, although the dilatation had shown itself only when the intra-abdominal pressure had been lowered, namely after the birth of the child.

At 1 p.m. her temperature was still only 35·5° C. (95·8° F.), but her pulse was a little better. At 6 p.m. her temperature was 35·2° C. (95·4° F.) and her pulse rate 120. However, at 10 p.m. her temperature had risen to 36·3° C. (97·4° F.) and the pulse was 116 and her condition definitely improved. The pain in her chest was still severe, but she had two good motions during the day and passed flatus freely, but her urine was scanty and highly coloured.

On the third day she was still very distended and she had become jaundiced. Calomel 0·03 gramme (half a grain) was given every two hours for four doses. Her bowels acted well during the day, but the motions were pale.

For the first time the vaginal discharge was offensive and a bright red rash had appeared over her buttocks. She had been very drowsy during the afternoon and had become slightly "mental." From this time onward she gradually improved, although she had various complications. The distension got less and less and the rash which spread over the whole of her abdomen was proved to be due to the use of "Lysol" and on changing the antiseptic it disappeared. Her mental condition at first got worse, but then gradually improved. The vaginal discharge cleared up after two large ulcers on the vaginal walls had been cauterized with carbolic acid and on August 13 she was discharged from hospital.

The main features of the labour were:

1. It had lasted sixty-eight hours, namely from 3 p.m. on June 17 until 11 a.m. on June 20.

2. Vomiting had started during the night of June 17 to 18 and during the day of June 18 she had complained that anything she ate or drank was at once vomited. On her admittance into hospital at 9.30 p.m. on June 18 she vomited greenish fluid and the vomiting from that time onwards was nearly continuous until 9.45 p.m. on June 19, when it ceased after the birth of the child.

3. The quantity of the vomitus had gradually increased. It had started by being small in amount, but increased until at 3.30 p.m. on June 19 she vomited three hundred cubic centimetres (ten ounces) and at 4 p.m. two hundred and ten cubic centimetres (seven ounces) of brown fluid and at 5.30 p.m. two hundred and forty cubic centimetres (eight ounces) were vomited.

4. The character had altered from a greenish fluid to typical thick brown coffee-ground vomitus.

5. From 9.45 p.m. on June 19 until the birth of the child at 11 a.m. on June 20 she did not vomit. I consider that most probably this was due to the action of the morphine.

6. The abdomen had not appeared to be abnormally large and had been examined at each visit, but I must admit that I had palpated the abdomen with the idea of gauging the descent of the head. However, I think I would have noticed any undue distension. The intraabdominal pressure most probably had been too great to allow the stomach to dilate, but as soon as this was reduced by the birth of the child, the stomach had dilated.

7. Six hours after delivery her pulse had risen to 140, but she did not vomit again until 2.45 a.m. on June 21, that is fifteen hours after delivery. From this time her temperature began to fall and went down to 35·1° C. (95·2° F.) and her pulse was 120, indicating collapse in which she remained for over twelve hours.

*Comment.*

The vomiting which so frequently occurs during labour, was very pronounced in this patient and the amount of vomitus gradually increased and its consistency and character altered.

De Lee,[3] in referring to vomiting during labour, writes:

Many women vomit during labour. The old midwives say that sick labours are easy . . . When the head is passing through the *os* the great distension may cause reflex vomiting and some women vomit frequently during the second stage. Persistent vomiting at any stage should always be suspected and its cause sought for carefully.

In this case no cause for the vomiting could be found and perhaps it would have been better if I had dilated the cervix and delivered the patient or even done a Cæsarean section. The vomiting was not due to obstruction as her bowels had moved several times. In the case reported by Berkeley and Bonney[8] laparotomy and Cæsarean section were performed, as obstruction had been thought to have been the cause of the vomiting, but at operation no obstruction was found. The question might be raised whether incessant vomiting during labour was an indication for *accouchement forcé* or Cæsarean section. In this case, however, the condition of the mother was good and the fœtal heart normal and I considered that the mother's condition did not warrant interference. However, if I had known at that time that the vomiting might be the precursor of acute dilatation of the stomach with all its dangers and discomforts, then I think I would have interfered earlier. It occurred to me afterwards that the passage of a stomach tube and washing out of the stomach might have relieved the vomiting The question remains: Is incessant vomiting an indication for interference during labour and, if so, at what time should the *accoucheur* interfere?

The patient became slightly septic and perhaps it was during the incubation period of this sepsis that the relaxing of the stomach musculature was initiated. Frank[14] in 1908 stated that he considered that sepsis was the main cause of acute dilatation and Hellendall[10] was able to find some form of sepsis in all his patients and he thought that sepsis was the cause. However, I consider that the sepsis is only one of many predisposing factors in the causation of acute dilatation.

A very small amount of chloroform had been administered. The vomiting, however, had started thirty-six hours before this, consequently the vomiting could not have been caused by the chloroform. Many observers have stated that chloroform is one of the causes and perhaps it might have had some toxic effect on the musculature of the stomach in this case.

There had been no operative interference beyond the slight pull with forceps to help the head over the perineum. The condition was therefore not a postoperative acute dilatation.

It therefore appears from the above that this case was really one of acute dilatation of the stomach, occasioned by vomiting which was reflexly caused by labour and perhaps accentuated by slight sepsis and a little chloroform.

I have searched through a large amount of literature and can find only the following reports of cases of acute dilatation which have occurred during labour or the puerperium.

Some of the cases, as will be seen, were proved at *post mortem* examination to be due to some other complication and these I have left out in my summary.

### Summary of Cases.

1. Thomson's patient[7] had had six children. Vomiting occurred during labour. The child was born successfully and vomiting ceased. Six hours later continuous vomiting started with collapse and all the signs of acute dilatation. The stomach tube was passed and a litre and a half (three pints) of fluid were drawn off. Death occurred six hours later. Autopsy revealed acute peritonitis from ruptured ovarian abscess.

2. Davis's patient[11] was a *primipara*. She was twenty-three hours in labour, the low forceps operation was performed, labour was long and the patient exhausted. Next day the abdomen was very distended and the patient vomited brown fluid. Next day there was greater distension. Stomach lavage was carried out every four hours. The patient was discharged, cured, on the twenty-eighth day.

Kuschnir's patient[9] was a *primipara*. Spontaneous birth of twins occurred after five and a quarter hours of labour. On the evening of the first day following labour the abdomen was greatly distended and the patient began to vomit and the next day vomiting and distension were worse On the third day the stomach was washed out and Schnitzler's position was adopted. Improvement occurred. However, next day vomiting again started and the stomach tube was again passed, but the patient got very dyspnœic and died. Autopsy revealed occlusion of the duodenum by the superior mesenteric artery.

4. Audebert and Fournier's patient[29] was a *primipara*. Eclampsia occurred, the patient received one hundred and fifty cubic centimetres of chloroform. Premature labour occurred and a seven months' fœtus was born. Eighty-four hours later acute dilatation occurred. A stomach tube was passed and gastric lavage was carried out. Cure resulted.

5. Audebert and Fournier's patient[30] suffered from eclampsia. Chloroform was given and then streptococcal infection supervened. Acute dilatation of the stomach occurred. Stomach lavage was used. Death occurred from general infection.

6. Mereau's patient[31] was a *primipara*. Long labour with violent pains was ended by forceps and ten grammes of chloroform were used. Immediately after delivery acute dilatation occurred. Cure resulted.

7. Le Lorier's patient[32] had spontaneous delivery before term, no previous gastric history was present. Four days later acute dilatation occurred. Gastric lavage was used. Death occurred five days later from perforation of gastric ulcer.

8. Boquel's patient[33] had had six children. Transverse presentation with prolapse of cord was present. Chloroform and version were used. On the third day acute dilatation was immediately cured by the adoption of Schnitzler's position.

9. Gilles's patient[35] had had five children. A shoulder presented, version was performed and rupture of uterus occurred. Operation was undertaken under chloroform anæsthesia. Thirty-six hours later acute dilatation occurred. Cure was effected by adoption of the ventral decubitus.

10. Audebert's patient[36] was a *primipara*. Spontaneous delivery occurred. No chloroform was used, no history of gastric trouble was obtained. Half an hour after delivery acute dilatation occurred. Cure resulted after eleven days with treatment by gastric lavage.

11. Audebert's patient[37] was a *primipara*. She suffered from dyspepsia during pregnancy. Chloroform was used, the breech presented, artificial delivery of afterbirth was carried out. Acute dilatation occurred immediately afterwards. Cure was effected by adoption of the ventral decubitus.

12. Van der Perk's patient[12] was a *primipara*. Labour lasted thirty hours. The abdominal wall was excessively flabby, six hours later vomiting and distension occurred.

Patient lay on her right side. Many eructations of gas occurred. Cure resulted.

13. Williams's patient[39] was a healthy woman. Spontaneous labour occurred. One hour after labour she passed into a condition of profound shock and a few hours later vomited and her condition was diagnosed as acute dilatation. Minimal amount of chloroform was used. Cure resulted.

14. Berkeley and Bonney's patient[8] was a full-time woman with acute abdominal pain and distension of the abdomen which increased. The abdomen was opened and Cæsarean section was done. Twenty-four hours later the patient was worse and copious vomiting occurred. Transverse colostomy was done and stomach contents were evacuated. Cure resulted.

15. Ramos's patient[13] had had twelve children. Labour was short, lasting two and a half hours, and the placenta was removed manually without an anæsthetic. Four hours later vomiting occurred, it became continuous and later of a dark colour. Great collapse and distension with pain occurred. The knee chest position was adopted and gases rushed out and the abdominal distension disappeared like magic. Twelve hours later the patient was quite comfortable.

16. Bretteville-Jensen's patient[40] had had eleven children. Twins were born with little chloroform. Much hæmorrhage occurred. Next morning acute dilatation and collapse occurred. The stomach tube was used. Cure resulted.

17. De Bovis's patient[41] suffered from *placenta prævia* with much bleeding. Acute dilatation occurred. (He gave no details of this case, but most probably there was some operative interference.)

18. My patient, herein reported, was a *primipara*. Labour was long and copious vomiting occurred during labour. Forceps were used with very little chloroform. Very pronounced distension occurred immediately after birth of child. The placenta was removed manually. Slight sepsis occurred. Cure resulted.

To this series of eighteen cases I can add six cases which have followed Cæsarean section, but I am not quoting these in detail as I consider them to be purely postoperative. For the sake of reference the authors of these Cæsarean cases were: De Bovis,[41] Ziegler,[42] Davis,[43] Kuliga,[44] Boquel,[34] Audebert and Fournier.[38]

I have spoken to a large number of obstetricians and most of them can quote a case or cases following Cæsarean section.

*Review of Cases.*

Every acute dilatation in the above series had some connexion with labour or the puerperium and it is seen that this complication must not be forgotten.

Case I should not really be included, as death was due to peritonitis and the condition was not one of acute dilatation, but of paralysis of the walls of the stomach and intestines due to the peritonitis. Case V and Case VII can also be left out for the same reason and Case IX as the uterus ruptured and an operation was performed. Case XIV cannot be classed with labour as the operation was done before labour commenced. We are left with thirteen cases and in these one patient or 7·69% died. This is contrary to the general opinion that patients with acute dilatation of the stomach usually die. The reason for this is I think that the cause of the acute dilatation has not been found and that in many cases it was due to peritonitis.

These thirteen cases might be considered to be a typical series of obstetrical cases, but of course containing more abnormal conditions than usual. Therefore, we see that in any obstetrical condition the possibility of acute dilatation taking place must always be borne in mind. It is difficult from the above series to deduce any one factor which predisposes to the condition. Operative interference and chloroform are the commonest factors. Nine patients had chloroform and there was some operative interference in seven instances.

The list includes:

Spontaneous birth, four cases, X, XII, XIII, XV.
Low forceps operation, three cases, II. VI, XVIII
Twins, two cases, III, XVI.
Breech presentation, one case, XI.
Eclampsia, one case, IV.
*Placenta prævia*, one case, XVII.
Transverse presentation, one case, VIII.

From the above list of recorded cases it is seen that the percentage of abnormal births is very high and that there is no one common factor beyond that of pregnancy and labour. There is one point to notice and that is the efficacy of postural treatment and lavage of the stomach. In this series of thirteen cases nine patients were so treated and the one patient who died, improved at first with stomach lavage. In the ætiology the necessity for continuous stomach lavage will be explained.

**Ætiology.**

The ætiology of the condition is not properly understood at the present time. Many suggestions and suppositions have been brought forward in the numerous articles on the subject, but no really definite advance in the ætiology as a whole has been made.

*Predisposing Causes.*

Very numerous predisposing causes have been suggested and on going over a large number of cases. I found that acute dilatation had accompanied or arisen in numerous conditions. Osler and McCrae[17] give a list of predisposing causes which include:

1. Debilitating conditions, such as pneumonia, toxic states, anæmia *et cetera*.
2. Trauma.
3. Postanæsthetic and postoperative conditions.
4. Dietetic errors.
5. Deformities of the spine.

Campbell Thomson[23] in his book divides his cases into five groups:

1. Those in which dilatation occurred without any apparent cause and in which after death no other lesion was found.
2. Those in which after death some other lesion was found.
3. Those following surgical operation.
4. Those following indiscretions in diet.
5. Those in which dilatation followed injury.

These groups in reality are only a division into predisposing causes and are approximately the same as were given by Osler and McCrae.

The above conditions only predispose to or have been found associated with acute dilatation, but they do not explain why the stomach dilates and why the patient vomits such quantities of fluid and then collapses.

*General Remarks.*

In most of the textbooks on surgery and medicine the various theories of the ætiology are discussed. Shelton Horsley[25] gives a large *résumé* of the ætiology, but does not come to any definite conclusion. He, however, states that in obstetrical work, acute dilatation occasionally happens, but that it is generally when chloroform has been used. He does not explain how the chloroform causes it.

Da Costa[26] in his book also discusses only the hypotheses of its ætiology and I have been unable to find any literature which definitely explains the ætiology.

*Hypotheses.*

Robertson[24] has collected the various hypotheses and gives them under three headings:

1. An upset of the nervous mechanism of the stomach and intestines causing a paralytic ileus.
2. An altered secretory activity of the mucous membrane of the viscera involved.
3. A mechanical ileus caused by the superior mesenteric vessels.

With reference to the first Fraser[27] suggests the theory that there is a balance between the sympathetic and parasympathetic nerve supply to the stomach and intestines.

For some reason this balance can be upset and various conditions will result, such as pyloric spasm, dilatation of the colon *et cetera*. Whether this theory can be applied to acute dilatation of the stomach has to be worked out and proved.

I will try to explain that these three subdivisions really all belong to the same cycle, one subdivision really being the cause of another.

I think it will be best to take the three main symptoms of acute dilatation and try to explain their ætiology.

*The Vomiting and Dilatation of the Stomach.*

The unstripped muscle walls of the stomach for some unknown reason suddenly relax and the organ becomes dilated. This condition of the stomach has often been seen in *post mortem* examinations. Lewis[18] in 1917 showed that involuntary muscles are not dependent entirely on their closely associated network of nerve fibres for their power of movement, but that the fibres and even parts of cells possess the inherent property of rhythmic contraction without any demonstrable nerve control. They also showed that after the rhythmical contraction has ceased it can be again instituted either by touching the cell or by the addition of calcium. Gardiner[19] applied this work to acute dilatation of the stomach. He held that in all cases a history of previous stress can be obtained and that the muscle fibres have become tired and relaxed.

Now any stimulus applied to the stomach wall will make these fibres contract and the stomach will be emptied. However, the sphincteric entrance and exit will also contract under the same stimulus and the stomach will be unable to empty itself. If a stomach tube be introduced the walls are irritated, but the cardiac end of the stomach is kept patent and the walls contract and the stomach is emptied. Gardiner came to the conclusion that all involuntary muscle possesses the property to function normally for a time and then after a period of stress to assume a state of relaxation and then after a limited time to react to stimulation and to return to normal functioning.

In nearly all cases there had been a history either of stress such as vomiting, labour, operative interference or of some form of toxæmia or infection and therefore the actual functioning power of the muscle fibres has been upset and they have become tired and relaxed and dilatation results.

We therefore see that some factor, such as vomiting, which in labour is most probably a reflex phenomenon, or toxæmia, tires out the musculature of the stomach and it is no longer able to retract to normal stimuli and the stomach therefore relaxes and dilates.

*The Collapse.*

In a most interesting paper, Dragstedt[20] brings forward experimental results that show that a severe or fatal toxæmia can quite easily arise from an arterio-mesenteric obstruction to the duodenum. He considers that the stomach dilatation is the primary factor and that the toxic symptoms are dependent on the secondary duodenal occlusion. In other words, the stomach becomes dilated, pushes down the intestines which pull on the superior mesenteric artery and the duodenum becomes blocked. The result is what is called the "closed loop," namely the contents of this piece of intestine undergo decomposition and toxic substances are formed and absorbed which cause a toxæmia and all the signs of collapse take place. Wilkie[21] has applied these "closed loops" experiments in his work on the ætiology of acute appendicitis with the same results, namely of a very acute toxæmia and in many cases of a very rapid death. The collapse is therefore due to a toxæmia which is caused by the dilatation of the stomach pushing the intestines down and so constricting the duodenum.

*The Excessive Amount of Vomited Fluid.*

The patient vomits and the amount of vomit is gradually increased. The origin of this fluid has been worked out by Luckhardt, Palmer and Henn[22] They short-circuited various loops and parts of the intestinal canal and were able to demonstrate the results of injections of various substances on the secretion of gastric, duodenal and pancreatic juices. Dragstedt[20] used their methods but injected toxic substances which he had collected from "closed loops" and he found that these toxic substances are very active secretogogues and have very pronounced effect on the gastric and pancreatic juices. Dragstedt also made chemical examinations on the vomited fluid and found it to contain all secretions, namely duodenal juice, pancreatic juice and bile, but very little hydrochloric acid. The above results can be proved by the use of the stomach tube. The stomach is emptied and so for the time being the toxic material is removed and the patient improves. Relapse, however, may take place owing to the formation of more toxic substances and the

emptying will have to be repeated. The actions therefore of the retained products in the duodenum act as secretogogues and the excessive amount of fluid is formed in the stomach and vomited.

Briefly the ætiology can be summarized as:

1. Some predisposing factor tires out the musculature of the stomach.

2. The stomach becomes dilated, pushes down the intestines and the duodenum becomes shut off by the pull on the superior mesenteric artery.

3. Toxic substances which are formed in the occluded duodenum, are secretogogues and so the amount of gastric and duodenal secretion is tremendously increased.

4. Later with the formation of more toxic substances and their absorption collapse from toxæmia and even death may take place.

In cases following normal delivery I consider the acute dilatation of the stomach to be due to a sudden releasing of the intraabdominal pressure and so to duodenal constriction. But in many cases, if the history were carefully examined, some predisposing cause for this relaxation of the stomach muscles, such as mild sepsis, slight anæsthesia, vomiting, would most probably be found.

There are several interesting points which can be concluded from the above remarks.

1. That acute dilatation during labour and the puerperium is very rare except when there is some predisposing cause.

2. That excessive vomiting during labour should always be a signal for investigations and that the likelihood of acute dilatation of the stomach taking place should be borne in mind.

3. That incessant vomiting during labour, for no apparent reason, may be an indication for rapid termination of the labour.

4. That the mortality of this complication of labour is not high, one death among thirteen patients or 7·6%.

5. That the prognosis, provided the correct treatment of stomach lavage and posture is carried out, is much better than was previously thought to be the case.

### Conclusion.

In conclusion I think that obstetricians should be on the lookout for this distension following delivery and should realize that the prompt use of a stomach tube will in practically all cases effect immediate relief and cure.

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## THE SCHOLASTIC AND CHARACTER CLASSIFICATION OF MENTAL DEFECTIVES.

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IN a recent number of THE MEDICAL JOURNAL OF AUSTRALIA attention was drawn by me to the mental and physical peculiarities of mental deficiency.[1] The classification of mental defectives, however, may be approached from rather a different aspect, namely that of individual scholastic attainments. The teacher is only mildly interested for example in head size or in the cranial peculiarities of mongolism, but is vitally concerned with the problem of how to assess the intellectual capacity, character and general adaptability of each pupil. It must be remembered that there is no standard intelligence or character for any type of mental defect.

The problem of how to train our defectives most advantageously is a serious one, since great expense is entailed. Special teachers have to be provided and it is essential that they should not waste their time on children who will show no profit.

Whilst a certain amount of overlapping between the educational and mental hospital authorities is inevitable, it is obvious that every refinement in the assessment of intelligence will make more easy the task of deciding the type of special school a particular child should attend. A case in point is the choice between the Mental Hospital, Newcastle, New South Wales, and "Glenfield," the special school of the New South Wales Educational Authorities.

In order to meet the above requirements at the former institution the following procedure has been adopted. The child's mental age and intelligence quotient are assessed by the Binet-Simon method (Stanford revision) shortly after admission and annually thereafter. A copy of the answers is filed for future reference. These and other data are correlated on a separate sheet for each pupil and are so arranged that by serving for the whole school period, it is easy to tell at a glance what improvement if any has taken place (see accompanying form). Columns as will be seen in the figure, exist for reading and writing, arithmetic, coiled basketry, cane work, sewing, toy making and planning. The standards for these are of the New South Wales Education Department. In addition a column exists for assessing ward work as used by the American Association for the Study of the Feeble-Minded.

The correlation chart was adapted from that used in the South Australian Education Department, for a copy of which I was indebted to Dr. Gertrude Halley. Examples of this chart are seen in Charts I and II. It will be noted that particular attention is paid to the handicrafts for the reason that advanced intellectual accomplishments are quite beyond the capacity of the defective to assimilate.

A yearly record is also made of character. The form used for this purpose is shown in Chart III and was drawn after consultation with Miss Holden, who has had long experience of the children's behaviour traits. It might be easily modified to cover the investigation of any other characteristics.

CORRELATION OF CHRONOLOGICAL, MENTAL, SCHOOL AND OCCUPATIONAL AGES

NAME _____ DATE _____ NO. _____

| YEAR | I.Q. | CHRONOLOGICAL AGE | MENTAL AGE | PROPER GRADE FOR CHR. AGE | SCHOOL: Reading Writing | Arithmetic | Coiled Basketry | Cane Work | Sewing | Toy Making | Planning | OCCUPATIONAL: Ward Work And Habits | REMARKS. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 18 | 16 | | | | | | | | | | |
| | | 17 | 16 | | | | | | | | | | |
| | | 16 | 16 | H.S. | | | | | | | | Can Supervise and Plan. | |
| | | 15 | 15 | H.S. | | | | | | | | | |
| | | 14 | 14 | H.S. | | | | | | | | | |
| | | 13 | 13 | H.S. | | | | | | | | Uses Machinery. Cares for Animals. | |
| | | 12 | 12 | H.S. | | | | | | | | No supervision, Cannot plan. | |
| | | 11 | 11 | VI. | | | | | | | | Fairly complicated work with only occasional oversight. | |
| | | 10 | 10 | V. | | | | | | | | Good institution Helper. Routine work. | |
| | | 9 | 9 | IV. | | | | | | | | Heavier work. Scrubs, mends, lays bricks, bares for room. | |
| | | 8 | 8 | III. | | | | | | | | Errands, light work Makes bed. | |
| | | 7 | 7 | II. | | | | | | | | Little Errands. Dusts. | |
| | | 6 | 6 | I. | | | | | | | | Tasks of short duration Washes dishes. | |
| | | 5 | 5 | Kinder Garden | | | | | | | | Only the simplest tasks | |
| | | 4 | 4 | | | | | | | | | Tries to help. | |
| | | 3 | 3 | | | | | | | | | No work, plays little. | |
| | | 2 | 2 | | | | | | | | | Eats indiscriminately. | |

GROUP 1 _____, GROUP 2 ....., GROUP 3 -.-.-.-; GROUP 4 ----,

CORRELATION OF CHRONOLOGICAL, MENTAL, SCHOOL AND OCCUPATIONAL AGES

NAME F.G. DATE NO.

| YEAR | I.Q. | CHRONOLOGICAL AGE | MENTAL AGE | PROPER GRADE FOR CHR. AGE | SCHOOL: Reading Writing | SCHOOL: Arithmetic | OCCUPATIONAL: Coiled Basketry | OCCUPATIONAL: Cane Work | OCCUPATIONAL: Sewing | OCCUPATIONAL: Toy Making | OCCUPATIONAL: Planning | Ward Work And Habits | REMARKS. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 18 | 16 | | | | | | | | | Can Supervise and Plan. | |
| | | 17 | 16 | | | | | | | | | | |
| | | 16 | 16 | H.S. | | | | | | | | | |
| | | 15 | 15 | H.S. | | | | | | | | | |
| | | 14 | 14 | H.S. | | | | | | | | | |
| | | 13 | 13 | H.S. | | | | | | | | Uses Machinery. Cares for Animals. | |
| | | 12 | 12 | H.S. | | | | | | | | No supervision, Cannot plan. | |
| 1926 | 66 | 11 | 11 | VI. | | | | | | | | Fairly complicated work with only occasional oversight. | |
| | | 10 | 10 | V. | | | | | | | | Good institution Helper. | |
| | | 9 | 9 | IV. | | | | | | | | Routine work. Heavier work. Scrubs, mends, lays bricks, cares for room. | |
| 1923 | 72 | 8 | 8 | III. | | | | | | | | Errands, light work Makes Beds. | |
| | | 7 | 7 | II. | | | | | | | | Little Errands. Dusts. | |
| | | 6 | 6 | I. | | | | | | | | Tasks of short duration Washes dishes. | |
| | | 5 | 5 | Kinder-Garden | | | | | | | | Only the simplest tasks | |
| | | 4 | 4 | | | | | | | | | Tries to help. | |
| | | 3 | 3 | | | | | | | | | No work, plays little. | |
| | | 2 | 2 | | | | | | | | | Eats indiscriminately. | |

Chart of an Actual Patient, F.G. It will be noted that his yearly increase in intellectual efficiency is smaller than for the normal child. The handwork makes a distinctly better showing.

CORRELATION OF CHRONOLOGICAL, MENTAL, SCHOOL AND OCCUPATIONAL AGES

NAME C.R. DATE NO.

| YEAR | I.Q. | CHRONOLOGICAL AGE | MENTAL AGE | PROPER GRADE FOR CHR. AGE | SCHOOL: Reading Writing | SCHOOL: Arithmetic | OCCUPATIONAL: Coiled Basketry | OCCUPATIONAL: Cane Work | OCCUPATIONAL: Sewing | OCCUPATIONAL: Toy Making | OCCUPATIONAL: Planning | Ward Work And Habits | REMARKS. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| | | 18 | 16 | | | | | | | | | Can Supervise and Plan. | |
| | | 17 | 16 | | | | | | | | | | |
| | | 16 | 16 | H.S. | | | | | | | | | |
| | | 15 | 15 | H.S. | | | | | | | | | |
| | | 14 | 14 | H.S. | | | | | | | | | |
| | | 13 | 13 | H.S. | | | | | | | | Uses Machinery. Cares for Animals. | |
| | | 12 | 12 | H.S. | | | | | | | | No supervision, Cannot plan. | |
| | | 11 | 11 | VI. | | | | | | | | Fairly complicated work with only occasional oversight. | |
| | | 10 | 10 | V. | | | | | | | | Good institution Helper. | |
| | | 9 | 9 | IV. | | | | | | | | Routine work. Heavier work. Scrubs, mends, lays bricks, cares for room. | |
| 1927 | 38 | 8 | 8 | III. | | | | | | | | Errands, light work Makes beds. | |
| 1926 | 43 | 7 | 7 | II. | | | | | | | | Little Errands. Dusts. | |
| 1925 | 50 | 6 | 6 | I. | | | | | | | | Tasks of short duration Washes dishes. | |
| | | 5 | 5 | Kinder-Garden | | | | | | | | Only the simplest tasks | |
| | | 4 | 4 | | | | | | | | | Tries to help. | |
| | | 3 | 3 | | | | | | | | | No work, plays little. | |
| | | 2 | 2 | | | | | | | | | Eats indiscriminately. | |

PLASTICINE MODELLING

PAPER CUTTING

Chart of an Idiot. This patient in three years has shown no improvement. Further school training is considered not worth while in his case.

TABLE I.

ANALYSIS OF CHARACTER TRAITS IN SIXTY-FOUR MENTALLY DEFECTIVE CHILDREN ATTENDING SCHOOL AT MENTAL HOSPITAL, NEWCASTLE, ARRANGED IN GROUPS.

| Character Trait. | Group I.[1] | Group II.[2] | Group III.[3] | Group IV.[4] |
|---|---|---|---|---|
| Unselfish | 3 | 2 | 4 | 17 |
| Selfish | 4 | 5 | 3 | 5 |
| Indifferent | 6 | 6 | 6 | 3 |
| Helpful | 9 | 8 | 6 | 17 |
| Helpless | 1 | 2 | 0 | 0 |
| Indifferent | 3 | 3 | 7 | 8 |
| Friendly | 11 | 10 | 10 | 15 |
| Quarrelsome | 1 | 2 | 2 | 2 |
| Indifferent | 1 | 1 | 1 | 2 |
| Leader | 0 | 2 | 5 | 8 |
| Follower | 10 | 10 | 8 | 13 |
| Individualist | 3 | 1 | 0 | 4 |
| Seclusive | 2 | 0 | 0 | 5 |
| Gregarious | 11 | 12 | 13 | 20 |
| Individualist | 0 | 1 | 0 | 0 |
| Self-conscious | 4 | 4 | 3 | 9 |
| Self-display | 1 | 0 | 1 | 5 |
| Normal | 8 | 9 | 9 | 11 |
| Constructive | 2 | 3 | 5 | 10 |
| Destructive | 3 | 2 | 1 | 3 |
| Indifferent | 8 | 8 | 7 | 12 |
| Obedient | 7 | 9 | 5 | 13 |
| Disobedient | 0 | 0 | 3 | 2 |
| Indifferent | 6 | 4 | 5 | 9 |
| Responsible | 0 | 1 | 2 | 4 |
| Irresponsible | 3 | 3 | 4 | 8 |
| Indifferent | 10 | 9 | 7 | 13 |
| Initiative | 2 | 1 | 5 | 7 |
| Initiative lacking | 5 | 4 | 1 | 8 |
| Indifferent | 6 | 8 | 7 | 10 |
| Adaptability | 6 | 2 | 7 | 7 |
| Unadaptability | 2 | 3 | 0 | 3 |
| Indifferent | 5 | 8 | 6 | 15 |
| Suggestibility | 9 | 10 | 8 | 13 |
| Negativism | 2 | 0 | 0 | 5 |
| Indifferent | 2 | 3 | 5 | 7 |
| Concentration | 9 | 4 | 6 | 13 |
| Easily distracted | 4 | 1 | 3 | 6 |
| Indifferent | 0 | 8 | 4 | 6 |
| Good temper | 9 | 8 | 7 | 10 |
| Bad temper | 3 | 3 | 5 | 9 |
| Indifferent | 1 | 2 | 1 | 6 |
| Clean | 7 | 4 | 8 | 14 |
| Slovenly | 2 | 1 | 0 | 3 |
| Indifferent | 4 | 8 | 5 | 8 |
| Undue sex interest | 0 | 0 | 4 | 2 |
| No sex interest | 1 | 2 | 1 | 1 |
| Indifferent | 12 | 11 | 8 | 21 |

[1] = Boy and girl babies.
[2] = Boys and girls, primary.
[3] = Boys' handwork.
[4] = Girls' handwork.

In practice the above procedure has been found to simplify greatly the classification in the school and well repays the labour entailed. An analysis of the findings gives a picture of mental deficiency, unobtainable by observation of the single child. Although the present data are too small to dogmatize upon, in time some very interesting deductions should be drawn.

There are four teachers at the Mental Hospital, Newcastle, the children being divided into four groups. In order to give an idea as to the nature of the latter, an account of the ages and school tasks is appended together with a correlation chart (Chart I) showing the average findings in the class and an analysis of the character traits (see Table I). It is hoped that a better view of the school activities will be possible than a mere verbal description could supply and may be of utility.

The grades of intelligence possessed by the sixty-four children attending school are set out in Table II:

TABLE II.—SHOWING INTELLIGENCE QUOTIENTS OF PUPILS.

| Intelligence Quotient. | Boys. | Girls. |
|---|---|---|
| 100—91 | — | 1 |
| 90—81 | 5 | 1 |
| 80—71 | 1 | 6 |
| 70—61 | 4 | 4 |
| 60—51 | 3 | 8 |
| 50—41 | 3 | 12 |
| 40—31 | 5 | 6 |
| 30—21 | 5 | 4 |

**Groups of Pupils.**

The pupils are arranged in the following groups.

*Group I.*

Group I consists of babies, boys and girls. The chronological ages are from four to nine years, the

CHART III.

TYPE OF CHART USED IN NOTATING CHARACTER TRAITS.

Name.....

| Character Traits. | Year |
|---|---|
| Social Attitude— | |
| Selfish or unselfish | |
| Helpful or not | |
| Friendly, quarrelsome | |
| Leader, follower | |
| Seclusive or gregarious | |
| Meddlesome, destructive | |
| Obedience | |
| | |
| | |
| Personal Qualities— | |
| Sense of responsibility | |
| Initiative | |
| Adaptability | |
| Suggestibility | |
| Concentration | |
| Temper | |
| Cleanliness | |
| Interest in sexual matters | |
| Self-conscious | |
| Self-display | |

FORM USED IN ORDER TO SIMPLIFY THE ACTUAL RECORDING AND FILING OF THE BINET-SIMON TESTS. IT IS MUCH LESS BULKY AND EXPENSIVE THAN THE STANDARD PATTERN.

NAME AGE.

YEAR III.

I. Nose eyes mouth hair
II. Key penny knife watch penc
III. D.H. C. P.O.
IV. Sex V. Surname
VI. Rep. 6—7 Syl. 1 2 3

YEAR V.

I. Comp. of Weights 1 2 3
II. Red yellow blue green
III. Aesth. Comp. Upper Middle lower
IV. Chair doll.
Horse Pencil
Fork Table
V. Patience 1 2 3 Time
VI. Commissions 1 2 3

YEAR VII.

I. Fingers R. L. Both
II. Pict. D.H.
C.
P.O.
III. Rep. 5 Digits 1 2 3
IV. Ties B.K. Time Method
V. Gives dif. Fly and b.f.
St. and Egg
Wood and Gl.
VI. Copies Diamond A b c

YEAR IX.

I. Day of week month D. of M. Year
II. Weights a b c
III. Change 10—4 15—12 25—4
IV. Rep. 4 Digits backwards 1 2 3
V. Sents. 1.
2.
3.
VI. Rhymes a time
b time
c time

YEAR XII.

I. Vocab. Score Tot. Voc.
II. A. d
b. e
c.
III. Ball and Field
IV. Dis. Sen. a Time
b Time
c Time

M. AGE. I.Q.

YEAR IV.

I. Comp. Lines 1 2 3
II. Dis. Forms Cir. Sq. tr. O.E.
III. Count 4 pennies
IV. Copies Square 1 2 3
V. A b c
VI. Rep. 4 Digits 1 2 3

YEAR VI.

I. R. Hand L. Ear R. Eye.
II. Mist. pic. Eye mouth nose arms
III. Count 13 pennies.
IV. Compreh. 1.
2.
3.
V. Threepence penny shil. sixp.
VI. Rep. 16—18 Syl. a b c.

YEAR VIII.

I. Ball and Field
II. Counts 20.0 Time Errors.
III. Comp. a
b
c
IV. Sim. wood and coal
Apple and Peach
Iron and Silver
Ship and M. Car.
V. Def. Balloon
Tiger
Football
Soldier
VI. Vocabulary Score Tot. Voc.

YEAR X.

I. Vocabulary Score Tot. Voc.
II. Absurd A
b
c
d
e
III. Designs a b
IV. Reading and Rep. Mem. Time M.
V. Compreh. a
b
c
VI. 60 Words 1 2 3 4 5 6

YEAR XII—(*Continued*).

V. Fables a
b
c
d
e
VI. Rep. 5 Digits backwards 1 2 3

mental ages are from two years six months to four years four months. The number of children in the group is thirteen.

The subjects taught are:

Use of *Kindergarten* and Montessori material in order to learn colour, number and size.

Occupations such as drawing, paper cutting, threading beads, plasticine, paper tearing.

Physical work such as ball games, circle games, rhythm and marching.

Singing nursery rhymes, finger songs and simple songs.

Simple household duties are performed. In addition pictures are shown and stories are read in class. Excursions are made to the beach.

*Group II.*

Group II is the first primary and includes boys and girls. The chronological ages vary between six and thirteen and a half years, the mental ages between three and a half and six years. The number in the group is thirteen.

The subjects taught are:

Use of *Kindergarten* and Montessori materials for number, colour and form.

Paper work such as paper cutting and folding.

Sewing such as card sewing and running stitch.

Raffia work such as raffia twisting and basket making.

Plasticine modelling, drawing, singing, stories, games, marching, gardening, rhythm.

Household duties include sweeping, dusting, floor polishing *et cetera*.

*Group III.*

Group III is the second primary and handwork group. It contains boys. Their chronological ages are from eight to seventeen, the mental ages from six to thirteen. The number in the group is thirteen.

The subjects taught are:

School work such as elementary reading, writing and arithmetic.

Paper work such as paper cutting, folding and weaving.

Drawing such as free drawing, designing and measuring.

The use of "Meccano" material.

Basket work such as making of raffia baskets of all kinds and shapes.

Cane work such as the making of trays, *jardinières*, vases, cake baskets, "billy" covers, waste paper baskets.

Wood work such as cutting with the fretsaw wood bases for trays and baskets, animals and figures for toys. Making boxes for ferns *et cetera*.

Garden work such as the care of flowers and vegetables, clearing ground for lawn and beds.

Physical work such as rhythm, marching exercises, games, sports, walks and swimming.

House work such as sweeping, dusting, polishing, window cleaning *et cetera*.

Singing, telling stories and letter writing.

*Group IV.*

Group IV is the group of girls' handwork. The chronological ages vary from twelve to twenty-two years, the mental ages from four and a half to ten and a half years. The number in the group is twenty-five.

The subjects taught are:

Plain sewing such as cutting out, tacking, use of sewing machine, ordinary sewing stitches, button holes, tapes *et cetera*.

Fancy work such as the making of tray cloths, table cloths, doilies, runners *et cetera* and all sorts of stitches.

Basket work such as the making of work baskets, cake, fruit, nut, sweet baskets and waste paper baskets.

Cane work such as the making of trays, cake and sandwich baskets, vases, *jardinières*, waste paper baskets, "billy" covers *et cetera*.

Canvas work such as the making of purses, bags and kettle holders *et cetera*.

Paper work such as paper cutting, covering boxes, making friezes.

Gardening, the growing of flowers and vegetables.

Woodwork such as cutting bases for trays and baskets, animals and figures for toys with the fretsaw.

Singing songs and hymns for church service.

Physical culture, rhythm, folk dances, exercises, marching, games and sport, walks and swimming.

Letter writing and stories.

House work such as sweeping, dusting, polishing floors, cleaning windows *et cetera*.

**Summary.**

1. An account is given of the methods used in assessing intelligence and character at the Mental Hospital, Newcastle, and in the compilation of school reports.

2. A description is given of school work, intelligence standards and character traits of sixty-four mentally defective children.

**Acknowledgments.**

I desire to record my indebtedness to Miss Joan Holden and her staff for their assistance in the compilation of this report.

**Reference.**

J. Bostock: "Mental Deficiency: Its Mental and Physical Characteristics," THE MEDICAL JOURNAL OF AUSTRALIA, February 19, 1927, page 255.

## Reports of Cases.

### NARCOLEPSY: A CASE AND SOME COMMENTS.

By H. HASTINGS WILLIS, B.Sc., M.B., Ch.M. (Sydney),
*Medical Officer, Repatriation Department, New South Wales.*

NARCOLEPSY is a clinical condition sufficiently uncommon to warrant a record of the following case.

**Clinical History.**

C.65653, a single man, aged now twenty-nine years, was a motor mechanic; when at the age of twenty years he enlisted for active service. He had suffered from pneumonia when seven years of age and whooping cough and chicken-pox when aged fourteen years. He left Australia in July, 1918, and his records show that he suffered an attack of "pyrexia of unknown origin" on Salisbury Plains, England, in October, 1918, necessitating eight days' stay in hospital. He arrived in France after the Armistice and saw no actual fighting. Whilst at the base he suffered from *otitis media* for eleven days and he fixes the date of origin of his present trouble at about that same time as he recollects suffering about fifteen minor sensations of momentary quivering of the muscles of the body during the six months he spent in billets in France, November, 1918, to May, 1919. While returning to Australia during his passage through the tropics in August, 1919, he had several attacks of loss of control of his legs. Still thinking the attacks of no importance, he did not parade sick and on discharge from the army in September, 1919, claimed no disability.

Since his army service he has been occupied as a dairy farmer and an orchardist in the coastal region of South Queensland and Northern New South Wales until two years ago, when he returned to his former occupation of motor mechanic. He has spent the greater part of his life in the country and has enjoyed generally good health. There is no family history of epilepsy or nervous disease.

He claims to be suffering from two kinds of seizures: (i) "attacks of loss of control" and (ii) "sleepy turns," quite distinct in character and apparently unrelated to each other. Apart from these seizures he has felt quite well.

The attacks of loss of control he first noticed, as above mentioned, about eight years ago and they have occurred with varying frequency since, but have been, he thinks, more frequent in hot weather than in cold weather. During recent years they have been less frequent than formerly and, to use his own words, occur now only when excited. They are of sudden onset and he has no warning. He states that in an attack he shakes and quivers in all the muscles of his body, he seems to lose control of himself, his legs give way under him and he has to catch hold of some stable object, such as a chair or table to prevent himself from falling. The duration of the seizure is less than one minute, about fifteen seconds, he thinks and he maintains that he is quite conscious throughout it. Observers have noticed his face flushed, his eyes staring, his facial muscles twitching and that he has difficulty in articulating his words, if speaking. Immediately after the attack he can proceed with his usual work and feels quite well. He gives no warning sign or other indication of the onset of a seizure.

For a year this was the only variety of seizure which troubled him, but since 1920 he has been troubled by sleepy

turns which he has found a greater disability. These commence with a feeling of weariness and somnolence which increases until he passes off into apparently quite normal sleep. These "turns" come on without apparent cause, but are more prone to occur in hot weather, when he is resting in a quiet place, when he is driving a motor car or when he is travelling in trains or trams. He has ample warning and can ward off "the sleepy turn" for some time, but ultimately succumbs and the sleep which then ensues is more prolonged and less refreshing than if he gives way and goes off to sleep at once. The duration of the period of sleep has varied from a half-hour to two hours when he is undisturbed and he fancies that he is more fit for work afterwards if suddenly awakened by someone than if he is allowed to awaken himself. He has on occasion slept only a few seconds and when awakened felt quite refreshed. The desire to sleep often comes over him at inconvenient times and he has been known to fall asleep when eating his meal at table, when lying under a motor car effecting repairs, when working with a fellow mechanic at a bench and on one recent occasion when driving a motor car. On this occasion he had travelled alone on a country road for forty-five miles, when he was awakened by colliding with a stationary car. Apparently his foot had fallen off the accelerator and his car was going very slowly, so that no harm resulted. This may have been an ordinary case of "dozing at the wheel," a not uncommon cause of accidents on long country roads, but in this case it is reasonable to regard it as a "sleepy turn."

On physical examination he shows no signs of organic disease. Mentally he is intelligent and apparently normal, though somewhat "phlegmatic," due perhaps to his life having been spent very largely in agricultural districts. His demeanour and conversation did not suggest the possibility of hysteria and he impressed me as a truthful witness.

A Wassermann test gave no reaction. An X ray picture of the skull, taken to show any enlargements of the *sella turcica*, failed to reveal any abnormality. A radiological examination of the alimentary tract by means of a barium meal revealed no functional or organic abnormality of the stomach or duodenum, but the appendix shadow was broken in outline suggesting a pathological condition there. A glucose tolerance test revealed a fasting blood sugar (twelve hours after food) figure of 0·104% rising half an hour after the ingestion of fifty grammes of glucose to 0·152% and one hour after glucose to 0·156%, then falling one and a half hours after glucose to 0·122% and two hours after glucose to 0·09%. The urine before and after this test contained no sugar or diacetic acid. Ordinary clinical examination of the urine revealed a specific gravity of 1020, acid reaction, no albumin, no sugar and no casts or pus.

### Comments.

His symptoms are similar to those of a group of twenty patients whom Adie[1] recorded recently as suffering from a disease *sui generis*, identical with the condition described by Gelineau in 1880 under the name of narcolepsy as a neurosis distinct from epilepsy. They differ from a typical case as described by Adie in that the attacks of loss of control, called by Adie cataplexy, preceded in date of onset the sleep attacks, whereas the opposite is the rule in previously recorded cases. Moreover in the typical case a cataplectic attack is provoked by some definite emotional state such as laughter or anger, whereas in this patient the relationship to emotion is less definite and the attacks occur only when the patient is excited. It is fair to assume that they are now less frequent, because he has come to realize their relationship to excitement and so is consciously or subconsciously able partly to control them. Moreover as the cataplectic attacks are reactive to emotion, one would expect to find them few in persons of an "unemotional" temperament or perhaps even absent altogether and Adie himself mentions such a case of sleep attacks occurring without cataplexy at all.

In a differential diagnosis (i) *petit mal* with sleep as an epileptic equivalent and (ii) paraencephalitis must be considered. From *petit mal* the condition in this patient is distinguished by the absence of unconsciousness in the cataplectic attacks, by the non-appearance of any major manifestation of epilepsy, though the condition has existed for at least seven years, by the absence of any cause for epileptic seizures, by the absence of mental impairment and by the absence of a family history of epilepsy. It is difficult to make sure that he does not lose consciousness in a cataplectic attack, as a patient may have no recollection of an attack of *petit mal*. In this case a trained nurse who observed the patient in an attack was of the opinion that he did momentarily lose consciousness, but the man stoutly denies it and from his account of what has happened around him during such attacks, I believe his statement.

From paraencephalitis the differentiation is more difficult. By paraencephalitis I mean the late nervous manifestation of *encephalitis lethargica* as by parasyphilis we mean the late nervous manifestations of syphilis and I accept the view that *encephalitis lethargica* is an acute infection with its seat of election in the basal ganglia, subthalamic region and the mid-brain, followed frequently by a subacute inflammation in some part of these areas and later by chronic degenerative processes in such places as the *globus pallidus* or *substantia nigra* as in Parkinsonism. Douglas MacAlpine[2] records that in eight cases of Parkinsonism examined by him subacute inflammatory changes were present in all especially in the mid-brain, though the only group of cells constantly affected was that of the *substantia nigra*.

Adie reports two patients with associated cataplexy and sleep attacks following frank *encephalitis lethargica*. Both apparently recovered and he considers them as different to his twenty cases of "idiopathic narcolepsy" and labels them "symptomatic narcolepsy." In this connexion the case of C.65442, a married male, aged thirty-eight years, is of interest. When seen by me a few months ago, he was suffering from Parkinsonian rigidity of the left side of the face, right arm and right leg, which apparently commenced about two years ago. At this time his friends commented on his stiff slow movements and suggested that he should "shake himself up." He gave no history of frank *encephalitis lethargica*, nor of any infection likely to have been abortive encephalitis, but this is not unusual as the majority of the patients with Parkinsonism in my small experience have not given such a history, due no doubt to the fact that mainly those patients the causation of whose condition is undetermined, attribute their disability to war service. His father is said to have died of *paralysis agitans*, but the weight of medical evidence favours a post-encephalitic lesion rather than a familial degenerative process in this patient. His first indication of ill-health was seven years ago when he suffered from uncontrollable attacks of apparently normal sleep occurring without adequate cause when working at his office desk or when travelling in the train to or from work. These attacks have been less frequent of late years, but he still goes off to sleep occasionally when reading a book or when talking to someone. Here we have no cataplectic attacks, but sleep attacks which have become less troublesome, while Parkinsonism has developed.

Is it that both these above-recorded patients are suffering from paraencephalitis and that the initial acute encephalitis was mild and abortive, a subacute inflammation in the structures in the floor of the third ventricle causing the cataplectic and sleep attacks and a degeneration of the cells of the *substantia nigra* causing the Parkinsonism? I cannot rule out such a possibility, but the subject is one for study by those more experienced than myself.

### Acknowledgments.

My acknowledgments are due to the Repatriation Commission for permission to record these cases and to Dr. A. W. Campbell, Consultant Neurologist to the Department, for confirmation of the diagnoses. I may state that in a critical review of the symptoms recorded, the possibility of suggestive questioning by the examining medical officer can be excluded.

### References.

(1) W. J. Adie: "Idiopathic Narcolepsy: A Disease Sui Generis, with Remarks on the Mechanism of Sleep," *Brain*, Volume XLIX, September, 1926.

(2) Douglas McAlpine: "The Anatomo-Pathological Basis of the Parkinsonian Syndrome Following Epidemic Encephalitis," *Brain*, Volume XLIX, December, 1926.

## IDIOPATHIC NARCOLEPSY.

By JOHN BOSTOCK, M.B., B.S. (Lond.), D.P.M.,
*Brisbane.*

ANY disease the study of which promises to shed light on the inwardness of that universally experienced but still little understood entity, sleep, is worthy of special study. Recently Adie[1] has drawn attention to a series of forty cases, described under the heading idiopathic narcolepsy —a term first employed by Gelineau. The condition is chronic and stated to be unaffected by treatment, the onset is from the age of thirteen and a half to thirty-nine years. The characteristic features are periodic sleep attacks without cause and "cataplexy" on emotion. As a result of some emotion, usually laughter, the entire musculature of the body becomes limp and powerless, the patient sinking to the ground conscious, but unable to move. The latter attacks last for a brief period of one-half to two minutes, the former may last for some hours. Usually they are quite independent and distinct, but occasionally sleep follows cataplexy and whilst the depth of unconsciousness varies, the condition is one of true sleep.

Adie describes attacks following *encephalitis lethargica* which suggests that the cause is to be found in the basal nuclei and pituitary regions. In the differential diagnosis he points out that the existence of a definite exciting factor rules out epilepsy, the attacks are quite unlike pyknolepsy. Against hysteria is the fact that suggestion is useless in treatment and the mental make-up of the patient is not hysterical. *Lachschlag* shows a superficial resemblance, but there are no sleep attacks and unconsciousness arises on laughing heartily.

The great medico-legal importance of this disease is obvious since attacks may occur when a patient is driving a motor car or at work *et cetera*. Concerning its relation to the theory of sleep, Adie sees in it additional proof of Pavlow's deductions. He regards sleep as being due to the spread of inhibitions on the lines described by the latter in his classical experiments on the conditioned reflex. Whilst the conception of "inhibition" may not be entirely satisfying, few will dispute Adie's contention that idiopathic narcolepsy is associated with an undue fatiguability of the nerve cells, a peculiar kind of nervous activity allowing excessive response to emotional stimuli. The case now to be described, whilst illustrating the main features of the disease, suggests that the results of treatment are more hopeful than hitherto supposed.

### Clinical History.

T.C., aged forty-six years, a wood worker, was sent to my clinic at the Newcastle Hospital by Dr. J. Davies on March 4, 1927. As illustrating the unexpected consequence of newspaper publicity, it may be mentioned that the patient sought treatment after reading an abridged account of Adie's paper in a daily paper. He had correctly diagnosed his own condition!

The family history manifested no similar abnormalities. Inquiry into his previous health revealed that he had had a fracture of the skull at eight years of age from a cricket ball striking him in the "eye." When twenty-two, he received a blow on the head with a brick, but was not unconscious. Recovery from these was complete. Eighteen years ago he began to notice weakness in the right knee and then he became drowsy and stupid. Ever since he has been subject to sleep attacks and muscular weakness.

The sleep attacks last for ten minutes to two hours. He can "fight them" for four hours when they become irresistible. The average is three *per diem*. These are not affected by emotion. He is unable to find a cause for them. Their incidence prevents him from following his occupation.

In the attacks of muscular weakness he loses power in his whole body and jaws. He finds it difficult to explain the feeling. These attacks average one a day, but are less if he is quite quiet. They are brought on by excitement, often when he cracks a joke or tries to fire a gun at a moving target, for example a duck. The onset is sudden and duration is from ten seconds to two minutes.

Although of the heavy phlegmatic type he is mentally alert and does not give an impression of hysterical tendencies. He dreams occasionally on lying down at night, but the subject-matter is of no particular significance. He does not suffer from headaches, except when he has a cold. There appeared to be no psychic trauma or psychological reason for his disabilities. There was no vomiting. Physically he is well made, of excellent physique and shows no sign of acromegaly. Examination of the cranial nerves revealed no abnormality. The pupils were equal and reacted to light and accommodation. The optic discs were clear. No sensory disturbances were present.

Rombergism was not present and ataxia was absent; flexor plantar reflexes were amongst the other findings.

Ankle jerks and knee jerks were not elicited even on reinforcement.

A radiogram demonstrated a deep pituitary fossa, the posterior clinoid process being larger than usual. The anterior posterior width, however, is small. There was no evidence of pituitary enlargement from tumour.

The cardiac and respiratory systems were normal. Examination of the urine revealed a specific gravity of 1015, acid reaction, absence of albumin and of sugar. He complained of frequency of micturition. The Wassermann test yielded no reaction.

He was treated by the exhibition of increasing doses of caffeine, *liquor strychninæ* and opium and is now, July 12, 1927, receiving caffeine citrate 0·6 gramme (ten grains), *liquor strychninæ* 0·6 mil (ten minims) and tincture of opium one mil (fifteen minims) three times a day. Opium was used on account of its action in preventing neural fatigue.

The patient now states that he can ward off the sleep attacks all day and has not had a muscular attack for some weeks. Six weeks ago he stopped a runaway horse without being affected in the slightest by the excitement, an event which astonished him. He confesses to feeling much better than for eighteen years.

Whilst it is inadvisable to dogmatize on a result which may be temporarily caused by suggestion, it would be interesting to try the above combination in other cases. Unfortunately the rarity of the condition rather precludes the collection of a series and is the chief reason for publishing at this juncture.

### Summary.

This report includes:
1. A description of idiopathic narcolepsy.
2. Description of a typical case.
3. Report of the promising result of treatment by caffeine, strychnine and opium.

### Reference.

[1] W. J. Adie: "Idiopathic Narcolepsy: A Disease Sui Generis, with Remarks on the Mechanism of Sleep," *Brain*, September, 1926.

## DIFFUSE GASTRIC POLYPOSIS ACCOMPANYING CARCINOMA OF THE PANCREAS.

By G. R. CAMERON, M.B., B.S. (Melbourne),

AND

R. J. WRIGHT-SMITH, M.D., B.S. (Melbourne).
[*From the Walter and Eliza Hall Institute of Research, Melbourne.*]

DIFFUSE polyposis is so rare a condition that additional cases are worthy of careful description.

Brunn and Pearl[1] recently have given an admirable review of this lesion collecting from the literature eighty-four proven cases. They conclude that gastric polyposis may be either congenital in origin, the polyps emanating from epithelial fœtal *Anlagen* separated during development, which are later stimulated to growth by chronic inflammatory irritation (Wechselmann), or may arise on a purely inflammatory basis. They quote the work of

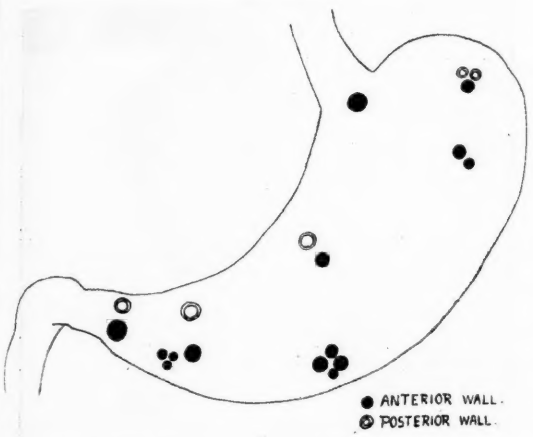


DIAGRAM SHOWING THE DISTRIBUTION OF POLYPI

FIGURE I.

Fibiger who was able to produce experimental polypoid mucosal growths in rats by feeding with a nematode. Gastric polyposis may appear either as distinct polypi, adenomatous in nature or as slightly elevated hypertrophic plaques, possibly telangiectatic in origin. The symptoms and physical signs are not characteristic, achylia being the most significant finding. X ray examination is a valuable diagnostic aid. Malignant degeneration occurred in 12% of the eighty-four cases collected.

### Clinical History.

E.J., male, aged sixty-nine years, old age pensioner, born in England, was admitted to the Melbourne Hospital on April 18, 1927, with the following history.

Two months before admission his appetite began to fail and he suffered from severe epigastric pain, commencing a few minute after meals and sometimes lasting for several hours, becoming more severe on taking food. The pain was not relieved by alkali powders. There was no vomiting. A fortnight later he became jaundiced and the pigmentation has persisted ever since. During this period he has lost 12·6 kilograms (two stone) in weight. His bowels have been irregular, constipation alternating with diarrhœa, the motions being dark-grey in colour. There has been no disturbance of micturition, although the urine has been very dark.

His past history was quite uneventful except for some vague gastric disturbance ("indigestion") thirty-five years ago. This continued over a period of three years. He has at no time taken an excess of alcohol.

On examination the patient was very wasted and deeply jaundiced. His temperature was 36·7°C (98° F.) his pulse rate 90 and his respiratory rate 18 in the minute. The pupils were regular, equal and reacted to light and accommodation. The tongue was dry and clean. He was edentulous. The urine had a specific gravity of 1025, contained much bile, was free from albumin and sugar. Microscopical examination revealed no abnormality.

The heart and lungs were normal. The systolic blood pressure was 126 millimetres of mercury, the diastolic 80 millimetres. The pulse was regular.

The abdomen was very distended, especially in the epigastrium, with an area of tenderness in the mid-line about five centimetres (two inches) above the umbilicus. There was no rigidity. Immediately below the right costal margin a mass moving with respiration could be palpated. The liver was not displaced. Movable dulness was present in both flanks and suprapubically, but no fluid thrill was obtained.

Examination of the central nervous system elicited no abnormality.

Loewi's adrenalin eye test yielded a reaction, but the diastatic content of the urine was twenty units only. X ray examination revealed a definite delay in emptying of the stomach. No intrinsic lesion was evident. Further investigations were impossible owing to the weak condition of the patient. He rapidly became worse and two days after admission following a severe hæmatemesis he died.

### Post Mortem Findings.

*Post mortem* examination was carried out eight hours after death. When the peritoneal cavity was opened, about three to four and a half litres (five to eight pints) of bile-stained fluid escaped. The peritoneum of the lower part of the abdomen contained many dilated veins and the omentum was adherent to the anterior abdominal wall.

The stomach weighed three hundred and sixty grammes (twelve ounces). It contained a large amount of old and recent blood clot, especially adherent to the free extremities of the polypi. Arising from the gastric mucosa were eighteen firm, cone-shaped polypi of varying size from a flattened type with a diameter at the base of four millimetres and a height of two millimetres to a long pedunculated type with a diameter of twenty millimetres at the base, ten millimetres at the apex and a height of thirty millimetres (see Figures I and II). There was no evidence of branching or of invasion of the submucosal and deeper layers of the gastric wall, the tumours being freely movable. The majority was situated on the anterior wall in the body along the greater curvature. On section, they manifested a central core of connective tissue with blood vessels and a definite thickening of the epithelial lining.

The remainder of the gastric mucosa was distinctly atrophic and the gastric walls hypertrophied to a moderate extent, this being especially pronounced at the pylorus. The blood vessels were not unduly prominent or thickened. The veins at the cardia were varicose.

The remainder of the intestinal canal was free from lesions.

Situated in the proximal portion of the head of the pancreas and involving the neck and adjacent portion of the body were nodules of hard, white tumour tissue. The duct of Wirsung was patent in its terminal four centimetres, but disappeared in the tumour mass. The tail of the pancreas was converted into a cystic structure with

FIGURE II.
Photograph of the Lesions on Stomach which has been opened along the lesser Curvature.

thick, fibroid, almost cartilaginous walls and containing whitish glairy fluid.

The common bile duct was compressed in its intrapancreatic course.

The peripancreatic glands along the superior margin and the cœliac lymphatic glands were infiltrated with malignant tissue.

The liver was enormously enlarged, weighing 8,760 grammes (ninety-two ounces), it was deeply bile-stained and very tough through great increase of fibrous stroma. On section thin, greenish-coloured bile gushed out from extremely dilated ducts. No secondary tumour nodules were present and the glands of the *porta hepatis* were free from lesion.

The gall bladder, cystic, hepatic and common bile ducts were enormously distended, with atrophic walls and recent adhesions to adjacent intestinal coils.

The myocardium was atrophied to a mild degree. The ascending and abdominal aorta manifested definite Mönckeberg's sclerosis.

The lungs were emphysematous and fibrotic and exhibited a terminal congestion and œdema throughout both lower lobes. A secondary malignant nodule was present in each lung.

The remaining organs in the thorax and abdomen presented no abnormality.

Microscopical examination of a typical polypus of the stomach revealed a complex, papillary-adenomatous structure. The lining epithelium was for the most part cuboidal, arranged in finger-like processes and plates several cells in thickness. Glands were numerous, varying considerably in size and forming definite cystic dilatations in the deeper layers of the *tunica mucosa*. They were lined by all gradations of cells from active mucus secreting columnar to flattened cuboidal or even pavement-like varieties. Chief and parietal cells were easily distinguished and there was much intracellular hyaline degeneration. Here and there cysts appeared to have been pushed between the layers of muscle in the *muscularis mucosæ*. There was a considerable amount of proliferation of the connective tissue stroma, all varieties of fibroblasts, but more especially the mature type, being easily distinguished. Lymphatic spaces were dilated and lymphocytes and plasma cells were fairly numerous. There was no evidence of any pathological change in the submucous, muscular and adventitial coats.

The polypi thus appear to belong to the "*polyadenomapolypeaux*" variety of Menetrier,[2][3] in that they were discrete, pedunculated, lobulated tumours bearing cysts and were probably the result of hyperplastic changes in the ducts of glands and the supporting connective tissue stroma.

The pancreas manifested a typical scirrhous carcinoma.

**Acknowledgment.**

We are indebted to Dr. Hume Turnbull for permission to make use of the ward notes.

**References.**

[1] H. Brunn and F. Pearl: "Diffuse Gastric Polyposis: Adeno-Papillomatosis Gastrica," *Surgery, Gynecology and Obstetrics*, Volume XLIII, 1926, page 559.

[2] P. Menetrier: "*Des polyadénomes gastriques et leurs rapports avec le cancer de l'estomac*," *Archives de physiologie normale et pathologique*, 1888, Series I, Volume XXXII, page 236.

[3] P. Menetrier and J. Clunet: "*Du polyadénoma gastrique à centre fibreaux et de son évolution cancéreuse*," *Bulletins et mémoires de la Société médicale des hôpiteaux de Paris*, 1907, Third Series, Volume XXIV, page 449.

## ERYTHEMA MULTIFORME BULLOSA HÆMORRHAGICA.[1]

By TERENCE C. BUTLER, M.R.C.S. (Eng.), L.R.C.P. (Lond.),
*Hobart.*

THE patient was a boy, aged twelve years, with a good family history and a history of no previous illness. He became ill and for six weeks manifested no definite symptoms. No cause could be assigned to his illness, until an erythematous rash of serpiginous type appeared round the face and chin and quickly spread over the whole body. On the third day bullæ appeared. They were mostly of a large size and at first contained clear fluid. Later the contents of the bullæ became hæmorrhagic and finally pustular. At this stage the temperature became elevated, but when the pustules were evacuated and antiseptic dressings were applied, the temperature was reduced considerably. The condition lasted for several months, the only drug which appeared to be of any assistance in treatment, being arsenic. Arsenic was administered for one year with frequent intervals and during that time many moderate attacks of a similar nature occurred. The condition finally cleared up completely in 1918 and the patient has been quite well ever since. The accompanying illustrations depict the appearance of the lesions.

[1] Read at a meeting of the Tasmanian Branch of the British Medical Association on June 14, 1927.

FIGURE I.
Showing *Erythema Multiforme Bullosa Hæmorrhagica* on Arms and Face.

FIGURE II.
Showing *Erythema Multiforme Bullosa Hæmorrhagica* on the Legs.

# CARCINOMA OF THE STOMACH WITH ASSOCIATED CEREBRAL MELANO-ENDOTHELIOMA AND EARLY MEDULLARY CARCINOMA OF A BRONCHUS.

By R. J. WRIGHT-SMITH, M.D., B.S. (Melbourne),
*Senior Resident Pathologist,*
*Melbourne Hospital.*
[*From the Walter and Eliza Hall Institute of Research.*]

## Clinical History.

W.T., male, aged forty years, fruitgrower, was admitted to the Melbourne Hospital on March 28, 1927. For the past five years he had had moderately severe attacks of localized pain in the left epigastrium, occurring half an hour after meals for two or three days at a time and relieved by food and alkali powders. During the last seven months he had been unable to work and latterly had been confined to bed. There was considerable anorexia and he had lost 12·6 kilograms (two stone) in weight. During the last three days he had vomited dark coloured material four or five times. There was no history of syphilitic infection or of alcoholism. At no time had there been any symptom suggestive of cerebral disturbance.

The patient was pale and wasted with œdema and pigmentation of the right leg below the knee. There were numerous areas of melanosis about one centimetre in diameter, scattered irregularly over the limbs. His temperature was 38° C. (100·4° F.), his pulse rate 96 and his respiratory rate 22 in the minute. The right pupil was larger than the left, but both reacted to light and accommodation. The tongue was atrophic and dry. The few remaining teeth were carious. The urine had a specific gravity of 1018, was acid in reaction, but there were no pathological changes discoverable by either chemical or microscopical examination. The heart was not enlarged, but a systolic bruit with maximum intensity at the apex was heard all over the præcordium. The systolic blood pressure was 130 millimetres and the diastolic 65 millimetres of mercury. The pulse was regular.

The lungs appeared normal to percussion and auscultation and there was no enlargement of the supraclavicular glands. Examination of the abdomen and central nervous system disclosed nothing abnormal. Rectal examination revealed no abnormality.

No abnormal cells were detected in the blood. The white blood corpuscles were 6,000 per cubic millimetre. The red blood corpuscles were 3,300,000 per cubic millimetre. Hæmoglobin value was 60% and the colour index was 0·9.

The fasting contents of the stomach as shown by a fractional test meal consisted of mucus, bile and blood; its volume was twelve cubic centimetres. There was no free hydrochloric acid and the combined acidity did not reach a value beyond 20. Emptying of the stomach was delayed. An opaque meal revealed pyloric obstruction with palpable and irregular *pars pylorica*, suggesting a carcinoma of the stomach.

The blood urea value was thirty milligrammes per hundred cubic centimetres of blood.

Nine days after admission the patient had a severe hæmatemesis. His blood was found to belong to Group IV (Moss's classification), but cross typing showed him to be incompatible with numerous donors of the same group. Transfusion was therefore not carried out. Six days later dulness and diminution of breath sounds with a coarse friction rub were discovered at the left base, persistent fine râles were found in the left interscapular region and diminution of breath sounds at the right base posteriorly. His condition steadily became worse and he died on the following day.

FIGURE I.
Adeno-carcinoma of Stomach. Microphotograph × 100 diameter.

## Post Mortem Findings.

*Post mortem* examination was conducted eight hours after death. The stomach was small and firmly adherent to the spleen, liver and pancreas. Projecting from the middle of the lesser curvature was a hard, papillary, fungating growth extending in a saddle-shaped fashion towards the cardia and pylorus. The edges were everted and very firm. On section a pinkish-grey infiltration could be seen, attaching itself to the inferior surface of the left lobe of the liver and the middle of the body of the pancreas. There was no evidence of recent hæmorrhage or of perforation. The peritoneal surface presented outlying nodules of growth. The glands of the lesser curvature, greater curvature and pylorus were grossly infiltrated with tumour tissue. Nothing abnormal was detected in the remainder of the alimentary canal.

The liver weighed 1,800 grammes (sixty-ounces). Its capsule was thickened with numerous adhesions round the gall bladder. On section pronounced toxic spoiling was discovered, but no evidence of tumour metastasis. The gall bladder showed a moderate cholesterosis. The pancreas and spleen apart from the changes described above were normal.

When the cranial cavity was opened, some clear fluid escaped. The cranial bones and *dura mater* appeared normal, but the pia-arachnoid was very adherent to the left anterior frontal lobe which was largely occupied by a deeply pigmented, hæmorrhagic, soft, roughly spherical tumour, with a maximum diameter of seven centimetres, extending posteriorly as far as the anterior horn of the left lateral ventricle, but having no connexion with the chorioid plexus. The tumour was deeply pigmented with melanin and contained cyst formation and old and recent hæmorrhage. The adjacent cerebral tissue was soft and pulpy. The remainder of the central nervous system and the ocular apparatus presented no abnormality. The heart weighed 360 grammes (twelve ounces). The parietal layer of the pericardium contained numerous pigmented secondary nodules from one to two centimetres in diameter, the visceral layer being clear, the myocardium pale, soft and not hypertrophied. The coronary arteries were normal, the heart valves free from lesions and competent. About

the middle of the postero-lateral wall of the right ventricle was a small pigmented tumour nodule involving the endocardium and myocardium. There was well-marked atheroma of the whole of the aorta.

The right lung weighed 1,320 grammes (forty-four ounces), the left 960 grammes (thirty-two ounces). The parietal pleura on both sides contained numerous pigmented secondary nodules one to three centimetres in diameter which, however, did not infiltrate the surrounding structures, the ribs and sternum appearing clear. Scattered throughout the lungs were numerous secondary melanotic nodules. Close to the hilum of the right lung and surrounding a bronchus of the second or third order was a white, spherical, hard, discrete mass, 2·5 centimetres in diameter. Secondary melanotic nodules were found in the tracheobronchial and preaortic lymphatic glands and in the posterior wall and pelvic portion of the peritoneum.

The remaining structures were free from lesions.

Microscopical section of the stomach (see Figure I) revealed a typical adenocarcinoma arising from the mucosal glands and infiltrating the deeper layers. The cerebral tumour (see Figure II) was highly cellular, its cells being extremely large, spherical or polyhedral in shape with copious foamy eosinophile cytoplasm. Nuclei were mainly peripheral, some active and in process of mitosis, others clear and vesicular, whilst some were flattened and crescentic. The cells were grouped around vascular spaces, actually forming the walls in many instances and occasionally invading the lumen. Groups of cells contained melanin pigment. There was very little stroma, that present tending to give rise to an alveolar arrangement. These appearances suggested a diagnosis of primary melano-endothelioma, probably of the hæmangio-endothelioma type.

The secondary nodules in the lungs, heart and pericardium and glands, manifested a similar arrangement of cells and blood vessels, but were much more pigmented.

FIGURE II.
Melano-endothelioma of Brain. Microphotograph × 150 diameter.

The tumour at the hilum of the right lung (see Figure III) was composed of a homogeneous spindle and oat-celled tissue. The cells showed scanty cytoplasm with elongated, deeply-staining, active nuclei. Stroma was scanty and for the most part finely granular with fairly numerous blood vessels, some appearing as clefts, others more highly organized. Scattered throughout the main tumour mass were clumps of incomplete tubules lined by large cubical and columnar cells with some mucoid degeneration. These appeared to be in continuity with the remainder of the tissue. In places a concentric arrangement of the cells was somewhat suggestive of imperfectly formed epithelial pearls. The appearances resemble those described by Barnard (*Journal of Pathology and Bacteriology*, Volume XXIX, 1926, page 241) in medullary carcinoma of bronchi simulating oat-celled sarcoma. On the other hand it is necessary to consider the possibility of metaplastic changes in connexion with the melanotic tumour tissue, for these tumours according to Dawson (*Edinburgh Medical Journal*, October, 1925) are especially prone to such changes, the same tumour presenting appearances of all three varieties of malignant growth.

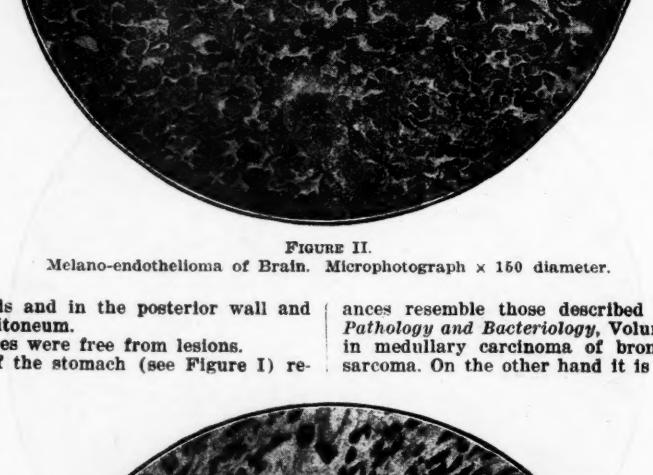

FIGURE III.
Tumour at the Hilus of the Right Lung. Microphotograph × 350 diameter.

**Acknowledgments.**

I am indebted to Dr. Hume Turnbull for permission to make use of the ward notes of this case and to Miss Cecil Maudsley for the microphotographs.

## Reviews.

### CERTIFIED MILK.

"THE proceedings of the Nineteenth and Twentieth Conferences of the American Association of Medical Milk Commissions in Conjunction with the Certified Milk Producers'

Association of America"[1] contains interesting and instructive papers of scientific importance, namely "Bacillus Abortus in Milk," "Hæmolytic Streptococci of Certified Milk" and "Foot and Mouth Disease."

The numerous discussions on improvements in dairy methods could profitably be read by milk producers in this country. They would undoubtedly find sufficient material to enable them to effect considerable improvements which would be beneficial both to themselves and also to the public.

According to the reports certified milk is gaining favour with the public and the very fact of the commissions being composed of medical men from the localities concerned, must tend to make the public appreciate a milk supply that is under constant expert supervision, and thus create a greater demand. The number of active commissions is apparently seventy-nine and the estimated number of certified milk farms in the United States and Canada is given as one hundred and forty-one, thus the movement has a large field for future extensions. The work must of necessity be arduous, but is more than justified by the improvement noted in both the dairy methods and the quality of the milk produced. The number of farms and dairies under supervision has also shown a gratifying increase. Publicity is taking a prominent part of the programmes and the reports indicate that the expense has been justified.

The commissions make a feature of herd testing for tuberculosis and according to the findings the strenuous exclusion of "reactors" is attended by good results.

It is unfortunate that the commissions do not employ a standard form for their yearly reports, as it is somewhat difficult to make comparisons. Some of these reports are particularly brief, but taking them as a whole there is a decided improvement noted over those of the previous years.

## EXANTHEMATA.

DR. FRANK V. G. SCHOLES, Medical Superintendent of the Infectious Diseases Hospital, Fairfield, Melbourne, is to be congratulated on the second edition of his book "Diphtheria, Measles, Scarlatina."[2] His work not only deals with the pathology, diagnosis, prognosis and treatment, but is a record of personal opinion and extensive experience in addition to a review of recent research and knowledge of those diseases.

The author insists that the diagnosis of diphtheria is to be decided primarily on clinical evidence. The finding of *Bacillus diphtheriæ* does not prove that a patient is suffering from diphtheria and failure to find it does not prove that he is not. In a review of over thirty thousand cases, valuable information is given as to the dosage of antitoxin. It is interesting to note that at Fairfield douching with warm normal saline solution is a routine measure. With regard to the operative treatment the indications for intubation and tracheotomy are extensively discussed, the former method being generally favoured. With regard to prevention application of antiseptics to the nose and throat did not prove efficacious. Removal of tonsils and possibly adenoids when diseased is a rational treatment when cultures yield the causative organism over a period of many weeks.

The Schick test is described in detail. In dealing with diphtheria contacts the recommendation is to give a prophylactic dose of antitoxin, if thought wise, to all those reacting to the Schick test and to those whose swabbings yield no organisms. In any case they should be immunized at once by injections of toxin-antitoxin mixture. For practical purposes all children under six should be regarded as susceptible and if they are to be protected they should be immunized. Older children, up to fifteen, should be subjected to the Schick test and if they react, they should be immunized.

In measles, on the second or third day of fever the appearance of Koplik's spots, small irregularly-shaped red spots with a tiny bluish-white point in the centre, render the diagnosis certain. In the absence of Koplik's spots, for example when the patient is seen too late, a pronounced form of the morbilliform variety of rubella (German measles) cannot be distinguished from measles.

All the evidence points to the fact that the germ of measles can live only a short time away from the human body and that the room after the removal of the patient need not be considered as a source of infection. The author considers that for practical purposes it can be said that the child in whom the exanthem has faded and the symptoms subsided, is not to be regarded as a source of danger. He has exposed many hundreds of susceptible children to desquamating convalescents, in some of whom the rash had not completely faded, and has never yet seen infection occur. Nine out of ten patients with measles do not come under the observation of the doctor until the rash is well out, if then. Immunization by serum of convalescents is not likely to be undertaken on a large scale, but may come into use for the protection of children in hospitals and babies' homes.

Scarlet fever is stated to be a disease due to a local and general action of a toxin elaborated by a streptococcus. In the Dick test we have a good general indication of relative susceptibility or immunity. However, in its present state it cannot be regarded as a safe guide in separating a community into susceptibles and non-susceptibles.

Results at Fairfield in the treatment of scarlet fever by doses of ten cubic centimetres of the concentrated serum of horses immunized by injections both of the toxin and *Streptococcus scarlatinæ* have been very encouraging and almost comparable with those obtained from diphtheria antitoxin patients treated reasonably early.

This work should prove useful not only to medical officers of infectious diseases hospitals, but can be commended to general practitioners.

## "THE CONSPIRACY OF SILENCE."

"THE REVOLT OF MODERN YOUTH" by Judge Ben B. Lindsey and Mr. Wainwright Evans is a book which should be in the hands of all who have the control of growing boys and girls.[1] Mr. Lindsey is a judge of the Juvenile and Family Court of Denver, United States of America. The sentiments expressed in the book are those of Judge Lindsey, but the actual writing has been done by Mr. Evans.

It is recognized by everybody that the modern youth of today enjoys much more freedom of thought and action than similar youths enjoyed when our grandparents were young. At the same time the available avenues of pleasure have increased to such an extent that they are regarded as the birthright of adolescents. The response of the individual to an appeal to his senses is the same now as it was fifty years ago, the endocrine organs of man have not altered. If there is a demand on the part of youth for greater liberty, in other words if there is a "revolt," it is a result of a change in civilization and not in the human make-up.

The work of a judge of the Denver Children's Court is apparently different to that obtaining in the Commonwealth of Australia. It would appear that the judge is consulted by parents and children alike in the privacy of his office in regard to any matter which is troubling them. The girl who has made a mistake in her sex life, and

[1] "Proceedings of the Nineteenth and Twentieth Conferences of the American Association of Medical Milk Commissions in Conjunction with the Certified Milk Producers' Association of America"; 1926. Brooklyn. Demy 8vo, pp. 345.

[2] "Diphtheria, Measles, Scarlatina," by Frank V. G. Scholes, M.D., B.S. (Melbourne), D.P.H. (Cambridge); Second Edition, Completely Rewritten and Enlarged; 1927. Melbourne: William Ramsay. Demy 8vo., pp. 321. Price: 21s. net.

[1] "The Revolt of Modern Youth," by Judge Ben B. Lindsey and Wainwright Evans; 1925. New York: Boni and Liveright; Sydney: Angus and Robertson, Limited. Demy 8vo., pp. 364. Price: 12s. 6d. net.

the small boy who has been annoying the police, come to the judge, the "Kids' Judge" as he is called, and the "Kids' Judge" is apparently a man with a big heart and an understanding mind. He holds that the freest discussion and the fullest possible teaching alone will prevent young people from making serious mistakes. The figures he gives are appalling. He has records in his court of 495 school girls who confessed to illicit sexual relations and of these one in twenty became pregnant. He estimates from this that 3,800 girls of high school age in Denver, a city with a population of 300,000, had had actual sex experience. He condemns in the strongest language the sanctimonious fool who brands as "immoral" every girl who has made a mistake and he shows from his records that sympathy and understanding with suitable instruction will in the vast majority of cases make a second mistake impossible. "The truth is—and every child knows it—that children think and act quite as logically and much more honestly, than adults; and that their mistakes come from their limited knowledge of facts, a limit which leads them frequently into reasoning from wrong or insufficient premises." His remedy is "sympathy, understanding and tolerance and a complete willingness to let young people order their own lives in the light of the facts."

Probably the most important factor in the building of character is self control. The poet has sung of sovereign power being possible only through "self reverence, self knowledge, self control." Reverence and control are concomitant and neither is likely to be obtained without knowledge. There has always been a "conspiracy of silence" in regard to matters sexual and medical practitioners will agree with every word Judge Lindsey utters in his desire to break the silence. Would that Australian practitioners would do more actual teaching in the homes of the people!

Judge Lindsey also discusses the attitude of modern youth to marriage. While he affirms his belief in the married state, he offers suggestions in regard to a recognition of unions which are not of so permanent a nature, such as the old Scotch custom of "handfasting"—trial marriage for a year and a day. "True marriage must be a free and voluntary partnership of two equal and independent personalities"—a condition more often obtained outside than in the bonds of wedlock. Of contraception he speaks fearlessly and at length. His championship of the rights of the unborn child is excellent and his appeal for proper treatment of the unmarried mother cannot fail to appeal.

It is not to be expected that all medical practitioners will give unqualified approval to Judge Lindsey's avowals in their entirety. Many will differ from him, most on minor points only and the more conservative perhaps on some of the larger issues, but all will admit that his book is an honest attempt to meet the needs of the world in its present state of civilization. This book carries many lessons for medical men and women and we commend them to read it.

## A HANDBOOK OF ELEMENTARY SURGERY.

THE second edition of "The House Surgeon's Vade-Mecum" by Dr. Russell Howard and Dr. Alan Perry is comprehensive and up to date.[1] It should prove of more value to graduates who have not had the opportunity of residency in a large hospital than to those who have, for the latter will naturally adopt the technique of their own school. All surgical emergencies are dealt with, besides the all-important subjects of asepsis, sterilization and general principles of wound treatment. Operations are but briefly outlined, the emphasis being placed upon preparation, theatre methods, after-treatment and complications. There is a useful chapter upon medico-legal matters and a sound discussion of the use of the principal anæsthetics. An appendix deals with examination for bacteria, preservation of specimens and the use of serum.

The volume is to be recommended to the young practitioner as it is both concise and sound.

[1] "The House Surgeon's Vade-Mecum," by Russell Howard, C.B.E., M.B., M.S. (Lond.), F.R.C.S. (Eng.), and Alan Perry, M.B., M.S. (Lond.), F.R.C.S. (Eng.); 1926. London: Edward Arnold and Co. Crown 8vo., pp. 528, illustrated. Price: 12s. 6d. net.

## THE ESSENTIALS OF DENTISTRY.

WE have received from Messrs. E. and S. Livingstone the "Outlines of Dental Science" in five volumes each of some one hundred and fifty pages, dealing with the subjects of dental anæsthesia, bacteriology, *materia medica*, radiography and prosthesis.[1, 2, 3, 4, 5] The books are intended for the use of students preparing for the examinations for the English diploma of Licentiate of Dental Surgery.

For revision purposes they are a handy synopsis and they would be valuable to students, when read, as the authors intend that they should, in conjunction with standard textbooks.

Two volumes, those on anæsthesia and bacteriology, stand in merit above the others. The volume on anæsthesia by Dr. G. F. Rawdon Smith, is a compact and useful book and in so far as general anæsthesia is concerned, the student need not consult a fuller textbook. The author deals with the theories of anæsthesia, administration, choice of anæsthetic, emergencies and medico-legal aspects.

The chapter on nitrous oxide is very complete and up to date. In Australia nitrous oxide has yielded place to local anæsthesia by the nerve block method. It is not usual to find dentists administering nitrous oxide gas as a routine practice. The surgical removal of teeth and most minor oral surgery can be done more easily and with less discomfort to the patient under local anæsthesia. When the operation cannot be done under local anæsthesia, most dentists prefer to place the patient in a hospital and to ask a medical practitioner to administer the anæsthetic usually by the intratracheal or intrapharyngeal method. Dr. Rawdon Smith does not devote much space to local anæsthesia.

This volume is an excellent book on general anæsthesia and is especially useful to those dentists who administer nitrous oxide.

The volume on dental bacteriology is by Dr. Ralph Broderick. The author who is lecturer in this subject at the University of Birmingham, has produced an essentially practical book and one that is equally useful to dental students and practitioners. He realizes that such a wide subject as bacteriology cannot be dealt with adequately in a book of only one hundred and fifty pages and recommends that Muir and Ritchie's "Manual of Bacteriology" be read in conjunction.

The value of the book lies in the convenient classification of the organisms with a brief note on their staining reactions, culture *et cetera*. Special features are the chapters on "Methods of Examination of Material from the Mouth" and "The Organisms found in the Mouth."

Dr. Broderick has pruned his subject to a minimum, yet has omitted nothing essential nor has he allowed his ideas to become obscure by brevity.

[1] "Outlines of Dental Science; Dental Anæsthesia," by G. F. Rawdon Smith, M.B., B.S. (London), M.D. (Liverpool); Volume I.; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 160, with illustrations. Price: 7s. 6d. net.

[2] "Outlines of Dental Science: Dental Bacteriology," by Ralph A. Broderick, D.S.O., M.C., M.B., Ch.B., M.D.S. (B'ham), L.D.S., R.C.S. (England); Volume II: 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 144, with illustrations. Price: 7s. 6d. net.

[3] "Outlines of Dental Science: Dental Materia Medica," by Prosper H. Marsden, M.Sc., Ph.C., F.C.S.; Volume III; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 155. Price: 7s. 6d. net.

[4] "Dental Prosthetics," by J. Douglas Logan, L.D.S. (Edinburgh); Volume IV; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 229, with illustrations. Price: 7s. 6d. net.

[5] "Dental Radiography," by Charles A. Clark, L.D.S., R.C.S.; Volume V; 1926. Edinburgh: E. and S. Livingstone. Crown 8vo., pp. 111, with illustrations. Price: 7s. 6d. net.

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## Pathologists.

THE subjects of bacteriology, parasitology and immunology occupy an important place in the book of modern medicine. In addition every medical practitioner is concerned with the ætiology and pathogenesis of disease and his practice is largely dependent on his ability to visualize the changes produced in the various organs and tissues by disease processes and to recognize the effect of these changes on the functions and chemical reactions of the organs and tissues. In order that he may be equipped with sufficient knowledge in these special branches of medicine, the curriculum for students contains courses in biochemistry, biophysics, pathology including immunology and bacteriology, as well as so-called clinical medicine and clinical surgery. It does not appear to be evident to those responsible for the effective teaching of pathology that the several divisions of this wide and important branch must be associated with research, but must be divorced from routine work. It is essential for the future development of preventive and curative medicine that a considerable body of skilled and learned practitioners should take up these specialties, in order that the general practitioner may obtain reliable information concerning the infecting organisms that can be recovered from the tissues and fluids of his patients and concerning the biological reactions of his patients' organism. These specialists must be trained for the work and they must have a reasonable assurance that their labours will command at least as good an income as that earned by the general practitioner. The first proposition to be established is that the clinical pathologist is indispensable in the modern medical team.

In the year 1905 a bacteriological laboratory was established in the Medical School of the University of Melbourne and the late R. J. Bull was appointed its director and lecturer in bacteriology. We have recently called attention to the expedient that Bull was forced to adopt in order to secure his teaching department. The University authorities failed to realize that the head of the department should be given facilities for conducting research work and that the remainder of his time should be devoted to teaching. Instead of this Bull had to render his laboratory self-supporting. He succeeded at a prohibitive price. By carrying out routine tests and other work known as clinical pathology he earned money for his laboratory, but much of the time that should have been given to teaching and research, was occupied in this ancillary activity. No one deplored the crippling effect of this enforced money-making more than Bull himself. There is no need to labour the point. It is self-evident to every member of the medical profession that teaching and research represent the highest and most important functions of a medical practitioner. We may therefore establish our second proposition that the University authorities should fix the remuneration of the professors and teachers in the Medical Schools at a level sufficiently high to render it unnecessary for them to undertake outside work and that ample provision should be made for the maintenance and equipment of the departments.

General medical practitioners do not always seek the aid of their colleagues working as specialists on their own account in private laboratories. On previous occasions we have complained that some men have all their laboratory work done at government institutions, no matter whether or not their patients can afford to pay private fees for this work. It is assumed that the patients are not charged for the examinations, for if that were done, it would be nothing short of deliberate robbery to retain the fee for work done by someone else. No honest practitioner would commit such an offence. The function of public laboratories must be clearly defined. Persons in poor circumstances whether treated in hospital or in private practice should not be deprived of the advantage of modern science and those in charge of government laboratories should receive a declaration with every specimen sent by a private practitioner that the patient cannot afford to have the work done by a private pathologist. Fees are charged for work done at the bacteriological laboratories of the University of Melbourne. This brings these laboratories into

competition with private practitioners, especially as the fees charged are lower than those usually charged in private practice. The effect of this competition has been to reduce the number of bacteriologists in private practice. This constitutes a further reason for the discontinuance of the routine laboratory examination within the Medical School. Experience elsewhere has proved that the supply will be governed by the demand, provided that just economic conditions are the order of the day. General practitioners must realize that if it is necessary to have examinations carried out for patients who pay them for their attendance, they have the duty to see that the pathologist who carries out the examinations, receives a commensurate fee. The alternative is distasteful to the medical profession. It is that this work is performed at private institutions by salaried pathologists. If this plan were extended to its logical conclusion, all medical practice would be conducted by servants of institutions or the State and the whole profession would become salaried. The third proposition may, therefore, be established that laboratory examinations carried out in government institutions, in hospitals and in other public establishments should be gratuitous and should be reserved for those unable to pay fees to private practitioners In all other circumstances the work should be conducteed by private pathologists.

## Current Comment.

### THE DIAGNOSIS OF TUMOURS OF BONE.

THE diagnostic difficulties associated with tumours of bone are at least as great as those encountered in connexion with any other group of lesions in the body. The difficulties are largely due to the fact that the full significance of the changes found on histological examination is not known. It is not surprising that on the one hand limbs have needlessly been sacrificed and that on the other delay fatal to the patient has sometimes been allowed to occur. If adequate care be taken, however, it is possible to make a correct diagnosis in a large percentage of cases. The monograph recently published by Anatole Kolodny on bone sarcomata should be extremely useful.[1] It is not only a comprehensive summary of present day knowledge of the subject, but it is a study based on a large amount of clinical material. Kolodny has described each variety of bone sarcoma and has discussed separately the clinical course and diagnosis of each. Diagnosis is the immediate concern of a practitioner confronted by a patient with an abnormal bone condition and it appeared that a useful purpose would be served if Kolodny's separate statements were brought together and discussed. It is recognized that a knowledge of the nature of disease and of the changes produced by it is essential to the diagnostician. For this reason there appears in another place in this issue a summary of Kolodny's interesting and important observations.

The diagnosis of bone sarcomata may be considered from three points of view, the clinical, the radiological and the microscopical. It should be unnecessary to point out that the taking of a careful and accurate history is of primary importance. In this respect bone sarcoma differs from no other pathological lesion. Kolodny holds that it is sometimes possible to make a correct diagnosis from the history and the radiological appearances without examination of the affected limb or of tissue removed for microscopical examination. While this is without doubt possible on occasions, it is a practice to be deprecated. Neither the radiologist nor the pathologist should, even in the most favourable circumstances, be regarded as a "penny in the slot machine." Both should see the patient in the presence of the practitioner in charge and if possible in the presence of each other. Investigation into the questions of trauma and of pain may yield useful information. Care must be taken in regard to a history of trauma. In discussing the ætiology of sarcomata in general, Kolodny states that it cannot be denied that trauma is frequently associated with the origin of bone sarcoma. Later on he is more cautious and expresses the opinion that trauma is an unreliable element in the history of a patient with a primary malignant tumour of bone. Most patients can and do recall an injury which they look upon as responsible for almost any lesion under consideration. For example, almost every woman with a lump in the breast will remember having received a knock or a blow on the affected spot. Kolodny asks the pertinent question as to what is to be considered as trauma. He asks whether the sudden tension of a muscle upon the bone into which it is inserted, is not to be considered as a trauma. According to the answer to this question the frequency of trauma in the clinical history will change. Two points call for emphasis in regard to trauma. In the first place the trauma supposed to be responsible is often sustained after the onset of the tumour. In the second place the history of trauma followed by pain and fever will more often than not lead to a diagnosis of osteomyelitis, when the real condition is an early Ewing's sarcoma. This occurs with such frequency that Kolodny advises that a diagnosis of a chronic inflammatory skeletal process should not be made until Ewing's sarcoma has been excluded. Chronic osteomyelitis is not likely to be confused with osteogenic sarcoma, but since the latter may be characterized by a sudden onset of pain, inflammation of the skin, fluctuation and fever.

[1] *Surgery, Gynecology and Obstetrics*, April, 1927, Part II.

it has been mistaken for acute or subacute osteomyelitis. Pain is generally a prominent symptom in bone sarcoma. It varies somewhat in the different types. In osteogenic sarcoma it precedes the appearance of a tumefaction by an interval which may vary from a few days to a few months. The pain appears when the tumour encroaches on the periosteum. It is very severe and of a boring or gnawing character. It is unusual for pain to be intermittent in character in osteogenic sarcoma except in the early stages. In Ewing's sarcoma, on the other hand, pain is usually intermittent. The attacks of pain in this condition are accompanied by fever. Each succeeding attack is of longer duration and of greater severity and the intervals between the attacks become shorter until pain becomes continuous. Kolodny does not discuss the reason for the difference in the pain in these two conditions. The pain in Ewing's sarcoma must be associated in some way with an inflammatory reaction. The febrile attacks are regarded by Kolodny as suggesting that the tumour is essentially a lesion of the bone marrow. The fact that the tumour is usually palpable when the pain becomes continuous, brings it into line with osteogenic sarcoma in which, as already mentioned, pain appears when the tumour reaches the periosteum. It is to be noted that pain is relieved by radiation therapy in malignant tumours of bone, but that in giant cell tumour, the benign tumour, the pain persists after radiation for a longer time. The view has been generally held that the diagnosis of myeloma is rendered easier by the occurrence of albumosuria and of Bence Jones proteins in the urine. Kolodny points out that these may be absent in a large percentage of cases, according to some observers up to 50% and that the presence of Bence Jones proteins has been recorded occasionally in carcinomatous skeletal metastases and in other medullary tumours.

The stage of development of a tumour and its structure influence its radiological appearances; hence it is impossible to state that such and such an appearance is typical of a certain type of growth. Kolodny holds that it is the custom at the present time to underestimate the importance of radiological examination in the diagnosis of bone tumours. It is doubtful whether this is so as far as Australia is concerned. At the same time he is probably correct when he states that clinicians do not recognize that radiography offers a method for frequent reexamination which is harmless to the patient but important to the medical attendant.

The radiological appearances of the different types of bone tumour are distinctive as a rule, but unfortunately variation occurs. The typical appearances may be described as follows. Osteogenic sarcomata have no definitely limited outline; since they remain beneath the periosteal investing capsule, the tumour often has a spindle shape; when the tumour is rapidly growing, the tumour is ossified only at the ends and is revealed in the skiagram by a wedge-shaped osteophyte at each end of the spindle, spoken of as lipping of the periosteum. It is unfortunate that non-sarcomatous lesions of the skeleton may sometimes present one or more of these features. A skiagram in an early case of Ewing's sarcoma manifests a diffuse involvement of the bone, due probably to a diffuse origin of the tumour throughout the Haversian system. Whereas in osteogenic sarcoma the shaft of the bone may sometimes be seen passing through the tumour grossly intact, it is manifest in a skiagram of Ewing's sarcoma that the bone is being slowly displaced by tumour. The skiagram in myeloma is characteristic and has the well-known mottled appearance. In giant cell tumour X ray examination reveals a bulky spherical shadow with a multicystic appearance as a result of the osseous trabeculation in the periphery. The shaft of the bone is absent and it appears as if the cortex is blown out from within the medullary cavity so as to form the thin bone shell which limits the tumour sharply from the surrounding soft tissues. Beyond the tumour the cortex and the periosteum are entirely unaffected and no periosteal lipping is seen.

Theoretically microscopical examination of a piece of the tumour removed for the purpose is an eminently suitable and satisfactory method of diagnosis. There are several objections to this course. In the first place there is always a risk that dissemination of tumour cells will result, that the growth of the tumour will be accelerated and that sepsis will occur. Kolodny also points out that in tumours previously irradiated it may be difficult to secure a sufficiently vital and representative piece of tumour to warrant a histological diagnosis. In the second place the appearances found are not always easy to interpret. Reference to the summary of histological appearances previously mentioned will show that there are numerous complicating factors, such for example as the lymphocytic infiltration which occurs in both osteogenic sarcoma and Ewing's sarcoma. Moreover when tissue is removed, all the removed tissue should be saved, because recurrent tufts may present secondary changes that will lead to error. Kolodny makes the significant statement that whereas surgeons are frequently anxious to have a section taken for examination, pathologists of experience recognize the dangers of such a course and advise that it shall not be done. Reference should be made to one means suggested by him for obviating the necessity for exploratory incision, at any rate in Ewing's sarcoma. He has pointed out that this tumour is particularly responsive to radiation and that the same is true of cellular osteogenic sarcoma. A therapeutic radiation test yields such satisfactory results that in Kolodny's opinion it should be used whenever the presence of Ewing's sarcoma is suspected. This statement must be read in conjunction with his pronouncement that "only a well-planned and carefully conducted analysis of the history and clinical picture together with a study of the findings of the radiological and pathological examination will reduce *ad minimum* the error in diagnosis." No one individual has either the necessary knowledge or experience to justify his acceptance of the responsibility of diagnosis in these conditions. By team work alone will the best results be achieved.

# Abstracts from Current Medical Literature.

## MORBID ANATOMY.

### Metastatic or Embolic Endometriosis.

JOHN A. SAMPSON (*The American Journal of Pathology*, March, 1927) has investigated the subject of metastatic or embolic endometriosis by making a histological study of uteri removed during the various stages of the menstrual cycle. The vessels of the uteri were injected with a suspension of bismuth. It was found that the uterine mucosa contains venous capillaries and large venous sinuses and that the latter empty into similar sinuses in the uterine wall. During menstruation blood escapes from the mucosal vessels into the surrounding tissues and pieces of the mucosa are set free in the extravasated blood. The author refers to the description by Meyer of the relationship between the invading endometrial tubules and the lymph vessels and of the way in which the former often follow these vessels and distort them without actually gaining access to the lumen of the vessel. He confirms Meyer's observation in regard to the extraendothelial course of endometrial tissue in a direct endometriosis, but believes that the vessels are usually venous sinuses and not lymphatic vessels. He also believes that the menstrual reaction of the endothelium alongside a vessel might cause rupture of the endothelial lining of the vessel and permit bits of endometrial tissue to escape into its lumen. He has not, however, been able definitely to prove this. Bits of uterine mucosa were found in the veins and sinuses of the walls of menstruating uteri. It was found that in some instances the appearances were due to artefacts. The author holds that if pieces of the uterine mucosa are found surrounded by blood in a vein or sinus or attached to the wall of the vessel by fibrin, it is evident that they reached this situation before the tissues were fixed. He found these conditions in three uteri removed during the menstrual period. He concludes that fragments of endometrial tissue are at times disseminated into the venous circulation during menstruation from the mucosa lining the uterine cavity and also from ectopic endometrial foci. Metastatic or embolic endometriosis arises from the implantation of these emboli in nearby veins.

### The Fat Content of the Thyreoid Gland.

R. H. JAFFÉ (*Archives of Pathology and Laboratory Medicine*, June, 1927) has investigated the fat content of the normal human thyreoid gland. Lipoid droplets are constantly found in the epithelial cells of the thyreoid after the first year of life and the author points out that little attention has so far been given to the question whether the lipoid droplets of the thyreoid have anything to do with the secretory activity of the gland. He has studied seventy thyreoid glands from persons varying in age from twenty-two hours to eighty-six years. Only fresh and apparently normal glands removed within a few hours after death were used. All the thyreoids except six contained fat; the six thyreoids were from children under the age of eleven months. There was little fat in the glands of nine children whose age was from six months to thirteen years. As a rule the glands rich in lipins were more common after the fortieth year than earlier, but in very old people a thyreoid gland containing little fat was occasionally found. On the other hand glands with much fat were not exceptional in persons under thirty years of age. In the epithelium fine dust-like granules appear between the nucleus and the internal margins of the cells. These granules give the colour reactions of neutral fat. Later they become more numerous, they grow larger and give the staining reactions of phosphatides. These droplets are ready to be discharged into the vesicular lumen. Varying numbers of droplets remain in the cells and once more change their reactions and cephalin mixtures are now predominating. These are finally discharged into the central lumen. After the cell has given off its fat content, the process of lipoid segregation begins again. In the colloid fat is found in three different forms. In the first place it appears as small droplets from one to two microns in diameter. This type of lipoid secretion is seen during the whole of life, starting with the first appearance of fat granules in the epithelial cells. The amount of fat in the colloid is parallel with that of the epithelium. In the second place large droplets from four to eight microns in diameter arrange themselves in mulberry-shaped groups and seem to be kept together by a thin capsule. They become more numerous after the fortieth year of life. They are excreted together with the lipoid content of a number of adjacent epithelial cells. In the third place foamy cells filled with fat globules are found in the colloid. These cells were found to occur after puberty in about half the specimens. The fat which they contain has the same property as the fat in the larger clusters. The stroma of the thyreoid often contains typical fat cells. They were found in about half the specimens examined. A constant relationship did not exist between the intensity ot the fat reaction of the plasma and the lipoid content of the epithelium. The presence of lipoid-carrying cells in the stroma indicates that some of the lipoid passes from the cells into the stroma. The author points out that both clinical and experimental data are available which in accordance with the microscopical observations point toward some relationship between the fat metabolism and the function of the thyreoid. He refers to the work of several observers in this connexion. He concludes that the factors which may influence the intensity of the lipoid excretion cannot yet be definitely determined. He holds that diseases do not seem to have any influence.

### Cholecystitis Due to Bacillus Welchii.

E. PEARCE GOULD and L. E. H. WHITBY (*The British Journal of Surgery*, April, 1927) report a case of cholecystitis due to *Bacillus welchii*. The previous history of the patient was good and the attack of cholecystitis was characterized by sudden onset with pain and vomiting. The gall bladder was removed at operation and the patient made an uninterrupted recovery. The contents of the gall bladder yielded pure culture of *Bacillus welchii*. The wall of the gall bladder manifested extensive ulceration of the mucous membrane and an acute inflammatory reaction with polymorphonuclear infiltration extending into the muscular layers. The mucous membrane was polypoid in many places and gave evidence of long standing inflammation. The author refers to the rarity of the condition and discusses the reasons for the absence of pronounced toxic symptoms before operation and for the uninterrupted progress of the patient. In the first place the infection was confined to the gall bladder and the focus could be removed completely at operation. In the second place the organisms in the interior of the gall bladder were in pure culture. In the third place the previous health of the patient was good. In the fourth place the strain of the organism was not one productive of exotoxins

### Chronic Gastric Ulcer and Pathological Changes in the Nerves of the Stomach.

HARALD OKKELS (*The American Journal of Pathology*, January, 1927) has subjected thirty-four specimens of chronic gastric ulcer to careful histological examination for the purpose of studying the pathological changes in the associated nerves. He finds that nerve elements are more abundant in association with chronic gastric ulcer than in normal stomachs. The number of nerve branches present and their size are increased. The individual fibres are not thicker; in other words, there is no hypertrophy. He also finds that nerve cells of the myogastric plexus are frequently present in the scar tissue of the ulcer and he points out that this observation is contrary to that of other observers. Nerves were found to be incarcerated in cicatricial tissue in half the specimens examined, in all but three there was an increase in perineural tissue caused by inflammation and in only one specimen was there any growth of interlacing connective tissue within the substance of the nerve. The changes may be designated perineuritis and arise by an ascending lymphangitis; the perineuritis extends continuously and not, as Perman believes, by jumping from one place to another leaving uninvolved tissue between. The author also describes certain proli-

ferative changes of the nerves which he designates central cicatrix neuromas. They may originate in the myogastric plexus and will then contain nerve cells or they may develope from the periarterial nerves and are then devoid of nerve cells. These nerve changes are of a secondary nature. In discussing his findings the author concludes that pain which is so prominent a feature of gastric ulcer, may be due to perineuritis and the involvement of the nerves in the contracting scar tissue. The neuromata may be a contributing cause to the chronicity of the ulcer.

## MORPHOLOGY.

### The Position of the Abdominal Viscera.

R. O. MOODY (*Journal of Anatomy*, January, 1927) states that as enteroptosis, visceroptosis, gastroptosis and coloptosis have been much discussed in the medical literature of the last twenty-five years and as there is still much difference of opinion as to what these terms signify and whether or not they should be applied to normal as well as to abnormal or pathological positions of the viscera, he undertook an investigation to determine the range of the normal position of the abdominal viscera and to establish a basis for a definite and accurate concept of what may rightly be called ptoses. Radiographs were taken of over a thousand students, men and women, not one of whom had a history of chronic gastric or intestinal trouble. This gave a fair cross-section of the physical types of healthy people that are found in most communities. The author's conclusions are that long stomachs with the lowest part of the greater curvature as far as fifteen centimetres (six inches) below the interiliac line, the lowest part of the lesser curvature as far as 7·5 centimetres (three inches) below the line, and the pylorus as far as five centimetres (two inches) below the line, are normal. Transverse colons dipping far into the true pelvis are normal. Caeca with the blind end low in the true pelvis are normal. Livers with the lower border 2·5 centimetres (one inch) or more below the interiliac line are normal. The ability of the stomach, the colon and the caecum to function normally is independent of their position. These facts justify the conclusion that enteroptosis, gastroptosis and coloptosis seldom or never exist as pathological conditions.

### Internal Genital Organs in the Fœtus.

AMY M. FLEMING (*Journal of Anatomy*, January, 1927) gives an account of normal internal genital organs of a female fœtus fifteen centimetres long, based on the examination of serial sections. No muscle fibres are found in the tubes, the walls of which are composed of primitive connective tissue. No trace of a median septum between portions of the uterine cavity developed from the two tubes is found. There was no projection of the *cervix uteri* into the vaginal canal. Glands corresponding to the prostate gland are found in the postero-lateral wall of the urethra, adjacent to the distal two millimetres of the vagina. The part of Gärtner's duct within the wall of the uterus and vagina is better preserved on the right than on the left side. There are no muscle fibres in the wall of the uterus, cervix or vagina, although they are present in the wall of the bladder, urethra and urogenital sinus. The sympathetic nerve fibres, supplying the internal genital organs, pass down the hypogastric nerves to the level of the cervix and then pass to the tubes, uterus, cervix and upper portion of the vagina by several branches which communicate with one another. These branches are intimately associated with branches of the uterine artery. Ganglionic cells are scattered along the hypogastric nerves and occur on each of their main branches, but only on that part of their course opposite the cervix and vagina. Within the wall of the genital tract no cells comparable in size, shape or staining reaction to the ganglionic cells found on the more proximal portions of the sympathetic nerve fibres are seen.

### The Transplantation of the Medulla Oblongata into the Brachial Region of the Cord in Amblystoma Embryos.

S. R. DE SWILER (*Journal of Comparative Neurology*, April 15, 1927) describes experiments which have been directed towards a clearer understanding of the forces involved in the normal morphogenesis of the central nervous system. A developmental influence of the medulla on the anterior end of the spinal cord was observed in experiments in which the medulla (caudal portion) and the first two spinal segments of the cord were substituted for the first five spinal segments. The experiments indicate that the inherent capacity for cellular proliferation is greater in the anterior end of the cord (caudal end of medulla to third spinal segment) than in the medulla itself when grafted to the brachial region. He shows that position alone does not regulate the number of cells that proliferate in the graft but that there are certain local regions of the central nervous system where proliferation of cells takes place from causes which are hereditary in nature, and that the activities of these regions are bound up with the development of the behaviour pattern.

### The Motor Muscle in the Cervical Cord of the Albino Rat at Birth.

A. W. ANGULO T. GONZÁLEZ (*Journal of Comparative Neurology*, April 15, 1927) finds that there is no segmental arrangement of the motor cells of the cervical cord of the albino rat at birth corresponding to the segmental arrangement of the spinal motor nerves. Nor are the motor cells homogeneously distributed throughout the grey substance, but instead form relatively well-defined groups if seen in cross-sections or nearly continuous columns if seen in longitudinal sections. Some of these columns are found throughout the entire length of the cervical cord; others are found only in the cervical enlargement. From the fifth cervical to the first basal segments there are more cell columns than above this level. This increase in columns is most noticeable in the latero-ventral part of the grey matter of the anterior horn. The so-called dorso-lateral and ventro-lateral groups are formed of several subgroups. It has been found that a degree of fusion exists among some of the columns described. In the study of the functional significance of these cell columns, their progressive embryological development must be taken into consideration, as it is believed that the progressive morphological differentiation of the musculature is associated with or perhaps dependent upon the anatomical differentiation of these columns. It seems to the writer than an accurate comparative study from the point of view of progressive embryological development is greatly needed to form the basis for complete understanding of the meaning of these cell columns—both their specialization and their functional activity.

### The Structure of Neurone Junctions in Sympathetic Ganglia.

O. W. TIEGS (*Australian Journal of Experimental Biology and Medical Science*, June 16, 1927) gives an account of an investigation which is an attempt to extend to the sympathetic system and its peripheral plexuses the observations, previously made on the spinal cord, of a direct neurone continuity at the neurone junctions. He finds that the predominant type of ending here is the same as that revealed in the spinal cord, that is the axone collaterals penetrate either into the body of the nerve cell or more commonly into its dendrites. They pass as neurofibrils towards the centre of the cell, where they anastomose; the anastomosing network appears to be the seat of the integration. In addition there occurs also a second type of ending in the form of a pericellular ramification of nerve fibres, closely enveloping the body of the cell. This is the only kind found in the frog's sympathetic trunk. The end ramification lies beneath the cell capsule, but it is not possible to state whether it penetrates into the protoplasm of the cell or whether it ends in close contact with the cell surface. It is pointed out that a discontinuity of the staining reaction constitutes no argument for discontinuity in the neurone junction. In the ganglia of a senescent, but not of a young human subject, the axones and dendrites may degenerate into large globules. Thus is brought about a gradual disruption of the nerve connexions within the senescent ganglion.

# Special Abstract.

## BONE SARCOMA.

Six years ago the American College of Surgeons recognized the complexity of the problem presented by the sarcomata in bones and founded the Registry of Bone Sarcoma. S. A. Codman, who initiated the scheme, was appointed first registrar. Anatole Kolodny, of the Department of Surgery of the State University of Iowa, has recently published a most important study of 700 cases in the Registry collection and has reviewed the literature on the subject.[1] His monograph is a formidable document of upwards of two hundred pages and is profusely illustrated with photomicrographs and reproductions of skiagrams. The investigation has not been confined to sarcomata; the benign giant cell tumour has been included, because "the relation of it to malignant bone tumour is still shrouded in mystery in the minds of the average clinician and pathologist."

### ÆTIOLOGY.

Kolodny points out that it cannot be denied that trauma is frequently associated with the origin of bone sarcoma. In regard to the relationship of trauma to the appearance of the tumour, he avoids "hypotheses of purely philosophical nature" and outlines the belief to which he is inclined after study of the Registry material. All osteogenic sarcomata may be arranged in a series in such a way that at one end is a mass resembling the callus seen after fracture and at the other a cellular, highly malignant looking osteogenic sarcoma. From this it is concluded that the development of osteogenic sarcoma is guided by the same principal laws of growth that are usually observed in the animal organism when in the stage of natural growth or when repair and regeneration are on hand. As long as the organism is in a state of physiological development, the cells grow without additional stimuli from the outside. When the organism reaches its adult stage no advanced growth is possible—"growth restraint" has occurred. The tissue cells in adult organisms do not lose their growth abilities, the latter from being kinetic become potential. No life would be possible if the potential growth ability of the tissue cells to multiply when repair is needed, were lost. Trauma with the consequent necessity of repair leads to a temporary change of the potential growth ability to kinetic, in other words growth restraint is temporarily eliminated in the traumatized region. In some instances in the presence of rare predisposing factors which are not understood, trauma may lead to a complete loss of growth restraint in the traumatized region. Kolodny points out that two facts must be considered in connexion with this view. In the first place when a tumour has appeared immediately after a trauma or when a primary tumour is noticed after a trauma not long before generalization of the disease is seen, it is not infrequently probable that the tumour has existed long before the trauma and that the latter has merely led to the discovery of it or to an exacerbation of its growth. In the second place the question not as to how often sarcoma has a trauma history, but how often a trauma is followed by sarcoma, adds to the discredit of the importance of trauma as an adjuvant ætiological factor in an organism predisposed to blastomatosis.

### CLASSIFICATION.

According to the classification accepted by the Registry Committee, bone lesions may be grouped under eight headings: (i) metastatic tumours primary in tissues other than bone, (ii) periosteal fibrosarcoma, (iii) osteogenic tumours, (*a*) benign, (*b*) malignant, (iv) inflammatory conditions, (v) benign giant cell tumours, (vi) angiomata, (*a*) benign, (*b*) malignant, (vii) Ewing's tumour, (viii) myeloma. Kolodny discusses only the primary sarcomatous bone tumours and, as previously mentioned, he includes the benign giant cell tumour. He divides all primary malignant bone tumours into the following groups: (i) osteogenic sarcoma, (ii) Ewing's sarcoma, (iii) myeloma, (iv) a group of unclassified sarcoma including among others such "near entities" as angio-endothelioma and extraperiosteal sarcoma.

### Osteogenic Sarcoma.

Ewing is responsible for the term osteogenic sarcoma. Osteogenic sarcoma includes mainly the lesions which have been known for long as "periosteal bone sarcoma." The true meaning of osteogenic sarcoma as understood by those who accept the Registry nomenclature, is a sarcomatous tumour derived from cells which, when duly differentiated, are known as osteoblasts. An osteogenic sarcoma is thus an osteoblastoma, somewhat as a carcinoma of the skin is an epithelioma. There is this difference, however, that while in the latter the epithelial cells are more or less typical and differentiated, in an osteogenic sarcoma various stages of development of osteoblasts can be seen, from a simple spindle cell to mucoid, cartilage and even to bone cell. A mistaken view is often held that an osteogenic sarcoma is a tumour producing bone. This is not so. Production of bone is merely a potential ability of tumour cells. Kolodny is at considerable pains to point out that the division of osteogenic sarcomata into periosteal, medullary and subperiosteal groups is valueless as far as clinical diagnosis goes. An osteogenic sarcoma is a diffuse process of the involved bone and there is no way to determine precisely the medullary or periosteal origin of the bone. To understand the capricious features characteristic of osteogenic sarcoma it is necessary to keep in mind the main factors which influence the gross picture, shape and consistency of these tumours. The foremost of these factors is the continuous aggression of the tumour elements and also the defensive protective measures of the involved bone against the growing tumour. The degree of differentiation of the tumour elements, the vascularity of the tumour and the regressive changes in it are factors greatly influencing the consistency of the tumour.

The growth and increase in the size of the tumour lead to more or less destruction of the bone in the involved area. Destruction of the bone is a condition without which there is no osteogenic sarcoma. The attempts of the tumour to extend are directed to two ways, one toward the outer aspect of the bone and the other along the medullary cavity. The latter growth is in the direction of least resistance. This explains the fact that the medullary cavity may be literally stuffed with tumour from end to end, while the skiagram indicates a tumour wholly limited to the area of involvement of the cortex and periosteal reaction. The periosteal reaction of a bone affected by osteogenic sarcoma consists in the formation of new bone apposed on the outer surface of the cortex of the involved bone. This apposition of new bone leads to a gradual thickening of the bone. The tumour gradually breaks its way through the old cortex and encroaches upon the newly apposed periosteal bone which it gradually replaces. Permeation rather than perforation of the cortex describes the pathological process. In this osteogenic sarcoma differs from Ewing's sarcoma in which the bone is gradually broken up and displaced by the tumour.

#### *Structure of Osteogenic Sarcoma.*

The histological picture of osteogenic sarcomata is most conspicuous and varied. There is no one type of osteogenic sarcoma. The cells from which these tumours are derived, are descendants of mesoblastic elements predestined embryologically to form bone, hence the cellular elements of osteogenic sarcoma have the potential ability to differentiate into osteoblasts. The apex of differentiation is not always reached. Something prevents the cell from climbing further up the ladder of differentiation and drives it to assume a lawless career. In almost every osteogenic sarcoma cells may be found which are arrested in their differentiation at various steps of the ladder. The histological picture is still further complicated by the great variety of intercellular substances; every stage may be encountered from simple fibrillation and hyalinization to true bone formation.

[1] *Surgery, Gynecology and Obstetrics*, April, 1927, Part II.

There are two types of cell which according to former teaching have occupied an important place in the histology of osteogenic sarcoma. Kolodny states most definitely that they do not really belong to this structure. One of these is the giant cell of the epulis type. The other is the round cell. It cannot be emphasized too strongly that if the leading cell of the tumour is a round cell, the tumour is not an osteogenic sarcoma. Erroneous statements in regard to the presence of round cells in osteogenic sarcoma are often due to the fact that the tumour has not been studied with sufficient care. The nucleus of a small spindle cell is small and hypochromatic and the cell borders are distinguished with difficulty, so that on cross section such cells easily simulate round cells. The most frequent type of cell in osteogenic sarcoma is the spindle cell. This is not surprising in view of the fact that the spindle cell is the standard type of cell participating in normal bone repair processes. The most frequently encountered spindle cell is of the small variety with a small hyperchromatic nucleus with indistinguishable cell borders. As the spindle cells grow they become larger, thicker, fuller and plumper and are known as large spindle cells. With further increase in size of the nucleus and of the amount of the cytoplasm the filaments become detached from the cell and the polyhedral cell results. As in other rapidly growing malignant tumours one of the main characteristic features of the tumour cell is hyperchromatism. Hyperchromatism is only of significance when demonstrated by good technique; it is then a more important sign of malignant disease than differentiation of the cells into fibres and bundles is a sign of benignity. The question of giant cells in bone tumours has given rise to much confusion. The distinction between tumour giant cells and giant cells of the epulis type is a morphological feature of prime importance. While giant cells of the epulis type may be met in most reparative processes of bone and in benign tumours and inflammatory processes, tumour giant cells are seen only in malignant tumours and especially in osteogenic sarcomata. The most frequent tumour giant cells are the so-called mononuclear giant cells, while polynuclear giant cells are seen chiefly in the rapidly growing osteogenic sarcomata. Kolodny here utters a warning that giant cells may be found in the benign granulation tissue of an infected wound and in a tumour which has been subjected to exploratory surgical operation. Giant cells of the epulis type are not infrequently seen in osteogenic sarcomata. They may be encountered in far advanced osteogenic sarcoma as a result of rapid and extensive bone absorption and they are not infrequent in necrosing and suppurating areas of such a tumour.

The chief morphological difference between osteogenic sarcoma and other tumours is in its intercellular substance. Kolodny holds that the study of the intercellular substance by the methods used in study of the cellular elements cannot lead to great enlightenment. Biochemical methods of investigation are urgently needed in the study of the former. The intercellular substance encountered in osteogenic sarcomata may be classified into five types, hyaline, osteoid, cartillaginous, myxomatous and osseous. Kolodny describes the successive changes in the intercellular substance and shows that ossification is the apex of differentiation which may be reached. It is the degree of ossification in osteogenic sarcoma that draws the line between osteoblastic and osteolytic forms. Kolodny believes that there are no pure osteoblastic or osteolytic osteogenic sarcomata. An osteolytic tumour, having destroyed much of the involved bone, usually manifests minute foci of newly formed bone sparsely scattered through the tumour. On the other hand in an osteoblastic tumour minute foci of cellular areas are found without any trace of bone formation. At the same time there is a decided difference in the consistency of an osteoblastic and an osteolytic osteogenic sarcoma. The whole difference is probably a chemical one. That it is not an anatomical one is seen from the rapid ossification which frequently occurs in an osteolytic osteogenic sarcoma after radiation.

According to morphological findings two types of ossification are encountered in osteogenic sarcoma, the metaplastic and the neoplastic. When under certain favourable conditions of the nature of which nothing is known, an organic compound added to the pre-existing fibrillar intercellular substance leads to homogenization and hyalinization of it without further calcification taking place, there results osseous tissue formed in a metaplastic way. The neoplastic ossification differs from the metaplastic chiefly in that in the former the fibrillar base for ossification is not pre-existing, but is formed in the process of ossification by way of cell proliferation. Focal proliferation of spindle tumour cells, arrangement in rows with fibrillation and hyalinization and finally calcification are the main stages of neoplastic ossification in osteogenic sarcoma. The terms neoplastic and metaplastic are based upon morphological and not upon biochemical data and it is not always possible to draw a strict line between metaplastic and neoplastic ossification.

Great importance has been ascribed to the discrimination in osteogenic sarcoma between bone formed by the tumour elements and that formed by the involved bone proper. The former is known as tumour bone and the latter as normal or reactive bone, that is bone formed as a result of a reactive inflammatory process of the involved bone due to the presence of the tumour. Kolodny points out that the crux of the argument regarding these two forms of bone concerns the presence in an osteogenic sarcoma of two kinds of newly formed bone, lamellary and trabeculated bone. He does not wish it to be understood that in his opinion tumour bone and reactive bone are entirely identical in every way; it is his belief that in the present state of knowledge there is no reliable way of distinguishing between them. The inelasticity of normal bone would prevent the growth and extension of an osteogenic sarcoma if it were not for the destructive process exerted by the tumour upon the involved bone. The absorption of bone under the aggression of an osteogenic sarcoma is accomplished in several ways. One way is the lacunary absorption of the bone, the way occurring in the normal process of bone resorption. In osteogenic sarcoma, however, the lacunæ are flatter and smaller than in normal absorption. Another means of bone destruction is canalization of the bone by advancing blood vessels. Extreme vascularization of the tumour mass may result in bone destruction as a result of direct pressure of the numerous pulsating blood channels on their bony capsule. Canalization with columns of tumour cells may also result in bone destruction.

The peculiarities of the vascular system constitute one of the characteristic features of osteogenic sarcoma. They are particularly rich in blood vessels, they are better supplied with blood vessels than normal bone. The only exception is found in osteogenic sarcoma with a purely cartilaginous matrix as a base. The blood vessels in osteogenic sarcoma are conspicuous in their anatomical structure and in their relationship to the tumour tissue. In the average pleomorphocellular osteogenic sarcoma numerous blood capillaries may be seen coursing along clefts between tumour cells. The tumour cells seem to carry the function of the endothelial lining of these blood channels. The same relation to the tumour may frequently be noticed in blood vessels of larger calibre. Areas can be seen in which cells of exactly the same morphological structure and appearance are lining blood vessels in one field and are indulging in bone formation in another. In still other places the endothelial lining of blood vessels seems normal, but the wall consists of a homogeneous mass exactly like osteoid or near osteoid tissue. The lining of a capillary with tumour cells raises the question of the origin of such a capillary. If the capillaries in these circumstances are formed according to general histological rules, they are formed by sprouting of a column of tumour cells, in other words there must be a most intimate relationship between normal endothelial cells and the tumour cells. Codman has suggested for these blood vessels the term tumour blood vessels. He sees in these peculiarities a proof of his view that the malignancy of bone sarcoma lies not in a certain kind of tumour cell, but in the intimate relationship of the cells to the blood vessels. He suggests that all osteogenic sarcomata are endotheliomata, because in his opinion the tumour cells spring from blood vessels as leaves from a tree. Kolodny thinks that there is probably more truth than error

in a belief that a malignant impulse imparted to the cells of a sarcoma lies much further back than in the origin of a malignant set of cells. It may reside in a disorder of the laws of growth.

Kolodny mentions specially the occurrence of a spontaneous lymphocytic infiltration. A lymphocytic infiltration occurs after radiation therapy and it is also seen in osteogenic sarcoma in which an exploratory incision has previously been made. A spontaneous lymphocytic infiltration is seen in only about 5% of all osteogenic sarcomata. From the fact that the lymphocytes are usually found on the periphery of the tumour it is concluded that they are not an integral part of it. Kolodny is opposed to the suggestion made by Ewing that all osteogenic sarcomata with a lymphocytic infiltration should be placed in a separate group.

The high malignancy of osteogenic sarcoma is due to a generalization of the tumour by metastatic deposits in other organs. No rules can be made as to the localization of the secondary tumour growths. They are known to occur in organs of the abdominal cavity, in the brain, in other bones and even in the skin. The most frequent, however, and the classical seat of a metastatic osteogenic sarcoma is the lungs. In a large majority of cases death results from pulmonary metastasis. A metastatic involvement of the regional lymphatic glands is rare. Frequently it is difficult to decide from the section alone whether the lymphatic gland is involved by metastasis. Occasionally an extensive hyalinization of the stroma of the enlarged gland is the only sign of metastatic involvement.

### Ewing's Sarcoma.

*Gross Anatomy.*

The gross anatomy of Ewing's sarcoma is characteristic and differs from that of osteogenic sarcoma. It favours the small bones of the extremities and the skull. In contrast to osteogenic sarcoma it involves the shaft of the bone much more extensively. The gross anatomical appearance in Ewing's sarcoma is mainly the result of the aggressiveness of the tumour cells, of the protective defensive measures of the affected bone and of the regressive changes in the tumour mass. It appears to begin simultaneously in numerous areas of the involved bone and these rapidly enlarge, become confluent and expand in all directions. At a very early stage of growth of the tumour the resemblance of the bone involvement to osteomyelitis is surprising. The resistance of the cortical bone is soon overcome by the tumour cells which rarefy and dissolve the bone diffusely from within the numerous Haversian canals. With the approach of the tumour to the outside of the bone the periosteum assumes its protective reaction. A shell of new bone appears as a reinforcement. The growth of the tumour, however, overtakes that of the new bone and finally the periosteum is perforated by the tumour and the latter infiltrates the soft tissues. The consistency of the tumour depends somewhat upon the stage of growth and the regressive changes which have taken place within it, and greatly upon its vascularity. While the blood supply does not reach such extremes as in osteogenic sarcoma, the degree of vascularity differs enough in various cases to influence the consistency of the tumour. Regressive changes are not uncommon and may result in cystic formations with yellow or red jelly-like contents.

*Structure.*

The histology of Ewing's sarcoma is simple. The typical cell is small and polyhedral with a round, oval or slightly elongated nucleus and with scant, clear, stainless cytoplasm. The nucleus is slightly stained with scattered granules which are distinguishable with difficulty or not distinguishable at all. The cytoplasm frequently forms a syncytium. No tumour giant cells are seen. A not uncommon finding is the arrangement of tumour cells about blood vessels, arterioles and capillaries. It has been suggested that the blood vessels present a structural unit of Ewing's sarcoma. Kolodny holds that while the tumour cells sometimes appear arranged like a mantle about cross sections of blood capillaries, there is no evidence to support the view that in this Ewing's sarcoma differs from other cellular sarcomata with the so-called perithelial or perivascular arrangement. Disintegration of tumour cells far from blood vessels leads to this appearance in any rapidly growing cellular tumour. In Ewing's sarcoma there is a complete absence of intercellular substance. The syncytial stainless cytoplasm of the tumour cells occasionally leads to the erroneous conclusion that the cells are embedded in an intercellular substance. The hyalinized blood vessel walls are another source of error in this regard. The blood vessel wall in tangential longitudinal section may appear as trabeculæ of hyalinized homogeneous connective tissue lined with rows of tumour cells.

The reaction of the tumour on the affected bone may complicate the histological picture. The extensive absorption of the shaft is accomplished without the help of giant cells. In sections there are frequently seen bone trabeculæ with typical morphological features of necrotic bone closely surrounded by tumour cells. Newly formed bone is of both the metaplastic and the neoplastic variety. Since the tumour cells in Ewing's sarcoma do not form any intercellular substance, this bone is regarded as a product of a regenerative process of the affected bone. The admixture of plasma cells is a feature which may complicate the histological picture. This fact suggests the question of the relationship between Ewing's sarcoma and a plasma cell myeloma. This question in Kolodny's opinion must be left in abeyance on account of the general lack of knowledge of the origin of myelomata. Occasionally a lymphocytic infiltration is seen in Ewing's sarcoma as in osteogenic sarcoma. This is situated in the periphery of the tumour and may be indistinguishable from inflammation in the bone.

Generalization of Ewing's sarcoma takes place by metastasis through the blood and lymph stream, but through the latter more frequently than in osteogenic sarcoma. Metastases are apt to occur in other bones of the skeleton, particularly in the skull. In the metastatic foci all the characteristic features of Ewing's sarcoma are repeated, especially the diffuse bone absorption. True bone formation does not occur in metastases from Ewing's sarcoma. This is important proof that the origin and physiology of the tumour cell in Ewing's sarcoma is different from that of osteogenic sarcoma.

Kolodny discusses the confusion by some observers of angio-endothelioma with Ewing's sarcoma and is emphatic that this confusion is at variance with all present-day evidence and knowledge. He also refers to the view that Ewing's sarcoma arises in the perivascular lymphatic endothelium, as suggested by Ewing. He holds that he is justified on the avialable evidence in suggesting the probable constitutional nature of the disease. He attaches significance to the fact that on the outskirts of Ewing's sarcoma there is evident an absence of blood-forming elements in the marrow. That Ewing's sarcoma is more than a simple bone tumour is evidenced by the diffuseness of involvement and the multiplicity of foci of origin in the same bone and also by the appearance of multiple tumours often very early in the course of the disease.

### Myeloma.

Myeloma is a rare condition. It is, however, in Kolodny's opinion of sufficiently frequent occurrence to warrant consideration in a study of primary malignant tumours of bone. The descriptions in the literature of its pathology and of its clinical course are widely divergent and Kolodny thinks that this will continue until more is learned of the normal anatomy of the hæmatopoietic system in general. Myelomata are found most frequently in the mid-portions of the long bones, especially the femur and the humerus, and in the flat bones of the skeleton. The tumour forms nodules ranging in size between that of a bean and that of an orange. The destruction of bone is less diffuse, has a more rapid course and is more circumscribed than that seen in Ewing's sarcoma. The gross specimen usually consists of a greatly thinned cortex of an expanded bone filled with an increased medullary substance, soft or firm, translucent or opaque, with little or no bone trabeculæ in it. The tumour mass is grey,

reddish grey or deep red according to the vascularity. Sometimes the vascularity is so great as to cause pulsation of the tumour. Extensive regressive changes are frequent.

*Structure.*

The histogenesis of myeloma is still the subject of much speculation. Tumours have been said to originate from each of the basic specific cellular elements, myelocytes, lymphocytes, mononucleated red blood cells. The majority of myelomata are composed of non-granulated mononucleated cells with opaque basophilic protoplasm, closely resembling plasma cells. The origin of these cells is much debated. The idea is being entertained that all types of myeloma are composed of cells merely representing various stages of anaplasia in a single cell. Ewing believes that the fact that the myelocytoma is of more active growth than the plasmocytoma suggests that a greater degree of anaplasia may lead a plasma cell to become a cell representing a myelocyte. The general trend of opinion favours the origin of the plasma cell from the lymphocyte or from the endothelium. Whatever the histogenesis of myeloma may be, four types of myeloma are classified, each with a different type of cell and possibly with a different clinical course. In order of frequency of occurrence these types are plasmocytomata, lymphocytomata, myelocytomata and erythroblastomata.

### Unclassified Sarcomata.

In a group of unclassified sarcomata Kolodny includes two conditions which are endowed with a typical anatomical structure and a characteristic clinical behaviour. These are angio-endotheliomata and extraperiosteal sarcoma.

*Angio-Endothelioma.*

Angio-endothelioma is a very rare tumour. The Registry series includes only two such tumours. Kolodny points out that no very extensive and definite conclusions as to their histology can or should be attempted. Such a course would not be safe theoretically and it is of no importance from the clinical point of view. Confusion has been occasioned in the literature by the failure of discrimination between primary angio-endothelioma and a secondary involvement of bone by an endothelioma in the neighbouring soft tissues. Another source of error arises from the fact that one of the leading features of angio-endothelioma is an abundant vascularity, hence confusion has occurred between this tumour and angioma and angiosarcoma.

*Extraperiosteal Sarcoma.*

By the term extraperiosteal sarcoma is understood a tumour which is intimately related to the periosteum, but is entirely extracortical, not invading or infiltrating the bone. This tumour arises from non-specific fibrous tissue which is incapable of producing osseous tissue. The leading histological picture of the tumour is that of a fibrosarcoma.

### Giant Cell Tumour.

As inferred by the name, giant cell sarcomata are lesions the histological structure of which is characterized by an abundance of giant cells. But not all tumours containing giant cells belong to this group. Three types of giant cell can be distinguished in pathological conditions. First come the giant cells which are the result of karyorrhexis in the cellular elements of the tumour. These cells are frequently observed in rapidly growing malignant tumours in which the division of the cytoplasm cannot keep pace with the closely following repeated division of the nucleus. These cells are a significant part of the parenchyma of the tumour. Following the lead of Mallory, Kolodny speaks of these as true tumour giant cells. A second type is the so-called foreign body giant cell. These occur in tuberculosis and in gummata. They are the Langhans type of giant cell with a peripheral or bipolar arrangement of their nuclei. The third type is represented by the giant cell of the "so-called giant cell sarcoma," called by Kolodny giant cell tumour. The nuclei are uniformly distributed throughout the central portion of the cell, they are of equal size and are completely separated from one another. Kolodny states that it is still insufficiently recognized that only lesions with giant cells of the third type are included under the term giant cell tumour.

Giant cell tumour is known by many different names. These include myeloid sarcoma, giant cell sarcoma, benign giant cell sarcoma of the epulis type, giant cell sarcoid, *osteitis fibrosa cystica*, hæmorrhagic osteomyelitis. The ætiology of giant cell tumour is intricately interwoven with the question as to whether the tumour is a neoplasm or a product of inflammation and repair. Over this question investigators are divided into two classes. Kolodny discusses this at considerable length. He holds that as the view that giant cell tumour is a true blastoma is supported by tradition, the burden of proof lies upon those who regard it as of inflammatory origin.

*Gross Anatomy.*

As in osteogenic sarcoma the gross anatomy of giant cell tumours depends largely upon the destructive and productive processes in the involved bone. The tumour tissue during its period of growth constantly destroys the bone while the periosteum lays down an advancing shell of new bone. Thus the giant cell tumour is an expansible but not an infiltrative or invasive lesion encapsulated in a bone shell. As the tumour expands, bony trabeculæ are left running in various directions in the "cystically" expanded bone shell. It is this structure that causes the characteristic "soap bubble" appearance in skiagrams. In a dried specimen the investing capsule of the giant cell tumour is seen to be furnished by the newly formed bone shell and the periosteum. The latter, while distinctly thicker and more vascular than on the adjoining uninvolved cortex, does not form, however, the abrupt elevation, the periosteal lipping which is so typical of osteogenic sarcoma. Unlike malignant tumour of bone, giant cell tumour is not characterized by early perforation of the periosteum. The periosteum continues to envelop the tumour long after the bony shell has disappeared. With the ageing of the tumour or after radiation therapy the tumour enters a cicatrizing phase. With the advancing differentiation of the tumour tissue fibrosis in all stages to fibrosis and cicatricial tissue may be seen.

*Structure.*

The histological structure of the typical giant cell tumour consists of two leading elements, the giant cells and the stroma. The stroma consists of numerous various sized blood spaces and exceedingly thin walled capillaries suspended in a very loosely woven net of spindle, round and polygonal cells with large vesicular nuclei. The absence of pleomorphism of the cells of the stroma is characteristic of the giant cell tumour. The giant cells are loosely embedded in the stroma. They are large opaque acidophile cells, resembling normal osteoblasts and containing numerous small oval nuclei uniformly shaped with a chromatin content larger than the nuclei of the stroma cells. The nuclei are grouped in the central portion of the cells. Kolodny refers to the much debated question of whether the giant cell is an integral part of the tumour or merely a side product called forth for scavenger work in a lesion loaded with decomposing material. He does not express his opinion about this question, but points out that, although the structure of the tumour suggests that the giant cell plays an important part in its composition, the giant cells when needed to do so, take up the function of scavengers and may be found filled with fatty detritus, remnants of blood cells, blood pigment and even small spicules of bone.

## British Medical Association News.

### SCIENTIFIC.

A MEETING OF THE TASMANIAN BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held in the Royal Society's rooms, Hobart, on June 14, 1927, DR. F. W. FAY, the Vice-President, in the chair.

### Erythema Multiforme Bullosa Hæmorrhagica.

DR. TERENCE C. BUTLER showed a patient who had suffered from *erythema multiforme bullosa hæmorrhagica* and read the history (see page 260).

### Exophthalmic Goitre.

Dr. Butler also showed a married woman, aged thirty-one years, with one child, whom he had seen for the first time on December 8, 1926. The patient had suffered from a goitre since she was fourteen years of age. The goitre had disappeared after a "nervous breakdown" when she was eighteen years of age, but had recurred twelve months later. The goitre had been removed three years previously at the Hobart General Hospital and she had been told that she was suffering from Graves's disease. Temporary partial improvement had taken place after the operation, but before very long the gland had again become enlarged. She had complained of exhaustion, continual nervousness, tremor, severe palpitation, headache, loss of weight and discomfort in the neck.

Examination had revealed that the patient was a small thin woman with a terrified expression, shaky movements and tremulous speech. The skin had not been moist. Tremor in the hands had been very pronounced and definite exophthalmos had been present. The pulse rate had been 135 in the minute and the weight 49·5 kilograms (seven stone twelve pounds). The left lobe of the thyreoid gland which had not been removed, had been very large. Mucus had been found exuding from the naso-pharynx and gingivitis had been present. The patient had been advised to have dental treatment and medical treatment had been undertaken. The patient had remained in about the same condition for about three weeks when all the symptoms had become exaggerated. She had begun to lose weight rapidly and to complain of epigastric pain and tenderness in the right iliac fossa. The patient's weight had been 45 kilograms (six stone twelve pounds). A chronically inflamed appendix had been removed in January, 1927, and immediate improvement in her general condition had taken place. On the second day after operation her pulse rate had fallen from 130 to 90 in the minute and had not become raised beyond this. In six weeks the patient had been quite normal, her weight had begun to increase, her pulse rate had been 80 and no tremor had been present. At the time of demonstration she was able to carry out her household duties without any effort and appeared to be perfectly well.

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A MEETING OF THE NEW SOUTH WALES BRANCH OF THE BRITISH MEDICAL ASSOCIATION was held at the Royal Prince Alfred Hospital, Camperdown, Sydney, on June 9, 1927, DR. F. BROWN CRAIG, the Acting President, in the chair. The meeting took the form of a series of demonstrations by the members of the honorary staff.

### Sir Charles Clubbe.

On the motion of DR. F. BROWN CRAIG, the Acting President, it was resolved:

> That the congratulations of the Association be conveyed to Sir Charles Clubbe on the honour recently conferred upon him by His Majesty the King.

### Artificial Pneumothorax.

DR. W. COTTER HARVEY showed a female patient, aged thirty-one years, whom he had seen for the first time in December, 1925. The patient's husband suffered from pulmonary tuberculosis. She had suffered from no previous illnesses, but had given a history of cough for two years. In October, 1925, she had been taken ill in Chicago with what was thought to be an influenzal attack. She had been told that she was suffering from early pulmonary tuberculosis and the skiagram shown by Dr. Harvey confirmed this. When he had seen her in December, 1925, the sputum had contained tubercle bacilli. Signs of early disease had been present at the left apex without toxæmia; her weight had been sixty-two kilograms (nine stone twelve pounds). She had been sent to the Queen Victoria Home at Thirlmere, but as she had not improved her husband had removed her and had decided to nurse her himself. Dr. Harvey had not seen her until the following September. Early in September, 1926, the patient had suffered from a febrile attack with increased cough and gastro-intestinal symptoms which had persisted for two weeks without improvement. She had been seen in consultation with Dr. E. H. Freidman, of Auburn. Signs of extensive left-sided disease with cavitation had been present and the right side had apparently been clear of disease. The evening temperature had been 38·9° C. (102° F.). Dr. Harvey had then recommended treatment by artificial pneumothorax and with this object in view the patient had been admitted to the Royal Prince Alfred Hospital. On October 7, 1926, the signs of the disease had been extensive with much pleural thickening and tubercle bacilli had been present in the sputum. Artificial pneumothorax had been produced. Before the operation the intrapleural pressure had been —12 on inspiration and —8 on expiration. After the introduction of 250 cubic centimetres of air these pressures had been —7 and —2. On October 18, 1926, after five introductions of air the collapse of the lung as estimated clinically had been good, the temperature 37·1° C. (99° F.) and the cough and sputum less. Before the fifth refilling the intrapleural pressure had been —5 on inspiration and —2 on expiration. After the introduction of 650 cubic centimetres of air these pressures had been 0 and +2. On October 25, 1926, the temperature had been normal for a week. X ray examination had revealed complete collapse except for a few adhesions at the apex. The patient's weight had been fifty kilograms (seven stone thirteen pounds). On December 2, 1926, the patient had been discharged. No tubercle bacilli had then been found in the sputum. Dr. Harvey pointed out that the patient had improved considerably. She was still having refills at regular intervals and her weight was sixty-five kilograms (ten stone four pounds).

Dr. Harvey also showed a man, aged fifty-four years, a tailor, whom he had first seen in October, 1925. The patient had complained of colds at irregular intervals for two years. He had suffered from hæmoptysis six weeks previously and his sputum had contained tubercle bacilli. Inquiry into his family history had yielded no information of value. He had been operated upon for a rodent ulcer of the face in 1919 by the late Herbert Maitland and the operation had been followed by pneumonia. On examination signs of infiltration had been present in the right interscapular region, but they had not been extensive. The patient had been sent to the Queen Victoria Home at Wentworth Falls where he had stayed for six months. He had gained 6·3 kilograms (one stone) in weight and on his discharge it had been thought that the tuberculous process in his lungs was arrested. He had returned to work for five weeks and had then suffered from an attack of influenza. On June 4, 1926, his wife had reported that he was spitting bright blood three or four times a day and that he had done so for one week. On the following day he had been admitted to the Royal Prince Alfred Hospital. In spite of intensive morphine treatment the patient had continued to cough up large quantities of bright blood (as much as three hundred cubic centimetres in a day). At this stage Dr. Harvey had undertaken the production of an artificial pneumothorax. The right side of the chest had been full of rhonchi and râles and the left side had seemed clear. Prior to the introduction of air the intrapleural pressure on the right side had been —8 on inspiration and —4 on expiration. After the introduction of 1,350 cubic centimetres of air these pressures had been —4 and +3. The patient had been quite comfortable throughout the operation. The bleeding had not been finally arrested until June 17, 1926, after several refillings. The patient had been discharged from hospital on July 21, 1926. At this time no tubercle bacilli had been present in the sputum and there had been no signs of involvement of the left lung. Dr. Harvey pointed out that the patient's general condition was much improved, he was keeping well and attended hospital every six weeks for a refilling.

Dr. Harvey's third patient was a girl, aged fifteen years, a laundress, whom he had seen for the first time on January 19, 1927. She had complained of cough for one

month. No sputum had been present; she had noticed a loss of weight for two months and had had night sweats for two weeks. She had stopped work for seven days. On examination the patient had been obviously ill with a hectic flush, a temperature of 39·2° C. (102·6° F.) and a pulse rate of 122. Her weight had been thirty-five kilograms (six stone two pounds). Diminished movement had been present on the left side with impaired note on percussion over the whole of the left side. The breath sounds had been tubular and weak at the apex and weak at the base. Numerous râles had been present over the whole of the left side, front and back. No abnormality had been discovered on the right side on clinical examination. Dr Harvey had recommended the patient for admission with a view to the production of artificial pneumothorax. He had regarded her infection as being acute in type and apparently still unilateral. This opinion had been confirmed by X ray examination. She had been admitted to hospital on January 25, 1927. The temperature had been 38·6° C. (101·6° F.) and the pulse rate 136. On January 28, artificial pneumothorax had been produced. Before the introduction of air the intrapleural pressure had been — 9 on inspiration and — 6 on expiration. After the introduction of 160 cubic centimetres of air these figures had been — 4 and 0. A considerable reaction had followed with a rise of temperature to 39·5° C. (103·2° F.), a running pulse and some cyanosis. The following day the patient had been somewhat better, but had still been cyanosed and complained of præcordial pain. The patient had gradually improved and for a while each refilling had been followed by a slight rise in temperature for one day. No signs had appeared in the right lung and good collapse of the left had been obtained. On March 27, 1927, the patient had been discharged to attend the out-patients' department. She was having refills every three weeks. Dr. Harvey explained that the patient was living quietly at home, doing nothing, but having no particular treatment. For the previous two months she had had no cough. She could be regarded as a brand saved from the burning, but the ultimate prognosis was not certain. With a two months' history such an extensive lesion indicated a rapidly spreading process—all caseation and no fibrosis. Girls at puberty as a rule did badly, but in his opinion no other treatment could have given such immediately satisfactory results.

### Cerebral Abscess.

DR. GARNET HALLORAN showed a patient who had suffered from frontal lobe abscess, and who had been operated upon by Professor F. P. Sandes. This case will be reported in full in a subsequent issue.

### Talipes Equino-Valgus.

DR. L. G. TEECE presented the case of a woman, aged thirty-two, on whom he had operated three years previously for paralytic *talipes equino-valgus*. As a substitute for the paralysed *tibialis anterior* he had transplanted the *peroneus longus* to the inner side of the foot. To supplement this a tenodesis had also been done, the *tibialis anterior* being implanted into the shaft of the tibia to hold the foot at right angles to the leg. The *peroneus longus* had acted most efficiently in its new position as a dorsiflexor, but the patient had asked to have the power of plantar flexion restored. He had therefore three weeks previously released the *tibialis anterior* from its attachment to the tibia. The *peroneus longus* was demonstrated acting as strongly as a dorsiflexor of the foot through its new attachment to the scaphoid bone on the inner aspect of the tarsus.

### Ununited Fracture of the Radius.

Dr. Teece also showed a patient who was suffering from ununited fracture of the middle of the radius, in which a sliding bone graft had first been performed without success. Subsequently a massive bone graft had been taken from the tibia and inserted into the radius. The considerable angular deformity which was present, had not been interfered with, since it was feared that the extensive freeing of the fragments of the radius which would have been necessary to restore the alignment of the limb, might diminish the chances of union in a patient who had already shown very defective powers of osteogenesis. The second graft had been successful, but six months later the patient had fractured the graft about its centre. Fortunately union of the fractured graft had occurred speedily. In spite of the angular deformity the patient had been able to return to his work as a fitter.

Dr. Teece's third patient was a child, aged three years, whose mother had brought it to the orthopædic department on account of a limp of one month's duration. On examination it was found that the right femur was bowed laterally and was 4·3 centrimetres (one and three-quarter inches) shorter than its fellow. The patient did not complain of pain. X ray examination had revealed expansion of the lower end of the femur with some thinning of the cortex. The radiologist was of opinion that the condition was either fibrocystic disease or else a hydatid cyst. The pathological tests for the diagnosis of hydatid disease had not yet been carried out.

### Congenital Bilateral Athetosis.

PROFESSOR W. S. DAWSON showed a male patient, aged forty-five years, who was suffering from congenital bilateral athetosis. The condition had first been noticed when the patient was eighteen months old. He had been unable to derive much benefit from education and had not been able to work. Professor Dawson pointed out that the patient displayed general athetoid movements of the face, of the limbs and to a lesser extent of the trunk. Good muscular power was present. The unusual features of the patient's condition were the bilateral athetosis, the absence of spasticity and the absence of knee and ankle jerks. The serum had not reacted to the Wassermann test.

### Cerebellar Gliosis.

DR. RALPH NOBLE presented a boy, sixteen years of age, who was reported to have been intelligent and healthy up till nine years of age. At that time he had fallen down the shaft of a mine fifty feet, but was not unconscious as a result. Some months later his school teacher had remarked that he was becoming mentally dull and the child complained of headaches and of "seeing rings before the eyes." During the previous five years the gait had become more and more unsteady and for the past three years he had had diplopia at times. The speech had become slow and difficult. Three years previously he had been admitted to Sydney Hospital where a diagnosis of cerebellar gliosis had been made. During the previous twelve months he had been unable to walk about unattended. In this period vomiting had occurred and this had recently become more frequent. The headaches were more severe.

He was able to read small print, but double papillœdema was present and this had been present when the patient was at Sydney Hospital. Recently the left side of the face had become weaker than the right and the left upper limb was not so strong as the right. There was nystagmus when the patient looked in either direction, but otherwise the eye movements were all accurate. The pupils were equal and reacted to light and accommodation. There was no diminution in the visual field, no loss of hearing was present and there was no past pointing, but there was some disturbance of deep sensibility. The reflexes generally were exaggerated, but no Babinski reflex was present. The patient's gait was very unstable and he needed support in order to walk. There was no loss of sensation to pain, light touch or heat or cold.

Dr. Noble concluded that the patient was suffering from cerebellar gliosis and the interest lay in its gradual onset and development following upon a trauma which was not accompanied by unconsciousness. Moreover there was no less of intellect in this old-standing condition.

### Post-Nasal Fibroma.

DR. A. O. DAVY showed a male patient, aged fourteen years, who complained of nasal obstruction of four months' duration. During this period the patient had suffered from three severe attacks of epistaxis. His hearing had been subnormal for several years, but had recently become worse.

The patient complained of neither headaches nor pain. Dr Davy pointed out that the patient had the so-call typical adenoid facies. The tonsils were enlarged and some cervical lymphadenitis was present. With the postnasal mirror a rounded, dark red, smooth tumour could be seen filling the postnasal space to about three-quarters of its extent. It appeared to be growing from the basi-sphenoid immediately behind the right choana. When it was touched with a probe, it felt firm and fixed and it bled very readily. From above its left upper border there protruded a pad of adenoids which was pressed over to the left side. On anterior rhinoscopy the tumour could be seen filling the posterior portion of the right side of the nose. Both tympanic membranes were retracted, the right more than the left and middle ear deafness which was present on both sides, was more pronounced on the right side.

The patient's serum had not reacted to the Wassermann test. A diagnosis of postnasal fibroma had been made.

Dr. Davy said that as it was impossible to exclude lymphosarcoma growing from the adenoid tissue and in view of the serious nature of a radical operation the patient was being treated by deep X ray therapy. No improvement was noticeable nine days after the first application. If no improvement were obtained after further application, the patient would be admitted to hospital for radical removal of the tumour.

### Hyperthyreoidism.

DR. F. S. HANSMAN reported three cases of hyperthyreoidism. This report will be published in full in a subsequent issue.

### Spina Bifida.

Acting on behalf of Professor A. E. Mills, DR. ANGEL MONEY showed a patient, aged twenty-three years, a slaughterman who had complained six years previously of "growing pains" in the back of both thighs and in the groins. Two months later he had become stiff and unable to bend. After a period of six weeks he had been attacked after doing some heavy work by severe pain extending down the back of both legs to the ankles. The pains had been present when the patient was at rest and and had often been worse at night. For a period of six years he had been unable to work and for a great deal of this time had been confined to bed. Wasting of the muscles of both lower extremities, but especially the left, had occurred. For a period of three years he had complained of numbness down the backs of both legs, in the feet and on the buttocks. Two years previously he had lost control of his sphincters. Eighteen months previously he had suffered from numerous "boils or ulcers" on the posterior aspect of the left thigh and six months previously from ulcers on the buttocks. At the time of demonstration he had two pressure sores on the buttocks, but these were almost healed. The patient had had a swelling in the lumbo-sacral region for some years.

Dr. Money pointed out that the patient was suffering from some dental caries, but that otherwise the alimentary system was normal. No abnormality could be detected in the respiratory, circulatory or urinary systems. Examination of the osseous system revealed a tense tender swelling in the lumbo-sacral area in the middle line. The swelling was fluctuant and pulsating and was about 7·5 centimetres (three inches) in diameter. Examination of the nervous system revealed impairment of sensation to heat, cold, touch and pain over the areas supplied by the fifth lumbar and the first, second, third and fourth sacral nerves. A light touch could be perceived over a small area supplied by the fifth lumbar nerve. The plantar reflex was flexor in type. The knee jerks were exaggerated. Patellar clonus and ankle clonus were absent. There was no alteration in sensation above the level of the fifth lumbar nerve. The patient walked with slight inversion of the left foot. He had neither rigidity nor pronounced weakness. Atrophy of the muscles was present in both legs, but more especially on the left side which measured 2·5 centimetres (one inch) less around the calf than the right. The patient suffered from incontinence of urine and fæces. X ray examination showed that the laminæ of the first and second segments of the sacrum were missing. This was regarded as a congenital abnormality.

### Encephalitis Lethargica.

DR. E. W. FAIRFAX showed two patients. The first patient was a man, aged forty years, an electroplater, who had been admitted to hospital on April 28, 1927. His wife had stated that he had lost his appetite and had complained of headache for one week. He had been drowsy and stupid for three days and had been unable to give coherent answers to questions. The day prior to admission he had had a convulsion, he had twitched all over and his face had become distorted. For a week prior to admission he had taken very little to eat or drink, his bowels had acted regularly and he had passed his urine twice a day. At the time of admission the patient had been found lying on his back with his eyes closed. He had not appeared to understand when told to put out his tongue or to open his eye and he had resented any attempt at examination or movement. The cardiac and alimentary systems had been normal. The pupils had been equal and had reacted sluggishly to light. He had moved his eyes from side to side when the eyelids were raised, but no abnormality had been detected in the other cranial nerves. Definite rigidity of the neck had been present and other findings had included exaggerated knee jerks, an active Achilles ankle jerk, plantar reflexes of the flexor type and Kernig's sign. For the first five days after admission the patient's temperature had been 37·8° C. (100° F.) and since then it had been subnormal, but had occasionally reached the normal level. For the first two days the pulse rate had been between 80 and 90 in the minute, for four days it had been between 60 and 70, for three weeks between 40 and 50 and since then between 60 and 70. On the day of admission lumbar puncture had been performed and thirty cubic centimetres of clear fluid had been withdrawn, it had flowed rapidly at first, the cell count had been 60 per cubic centimetre, lymphocytes predominating. It had been sterile on culture. No reaction had been obtained when the cerebro-spinal fluid was subjected to the Wassermann test.

Dr. Fairfax then described in detail the various observations that had been made during the patient's progress. These had included the performance of a Widal test to which no reaction had been obtained, the leucocyte count at the time being 18,000 per cubic millimetre. By May 2, 1927, it had been possible to rouse the patient and he had put out his tongue when told to do so. Incontinence of urine had still been present. By May 18 his mental condition had become brighter, he had opened his eyes and put out his tongue when told to do so, but he had been very irritable when disturbed. By May 22 he had been quite cheerful, he had talked incessantly, but quite irrelevantly. By June 7 he had been less excitable, could remember where he worked and where he lived, but not any recent events. Lumbar puncture had been performed on several occasions; that of June 1 had yielded fluid under increased pressure. It had contained lymphocytes, its glucose content had been 0·06% and the chlorides 0·7%.

The second patient shown by Dr. Fairfax was a man, aged fifty-two years, an estate agent, who had been admitted to hospital on February 26, 1927. Five days prior to admission the patient had complained of headache and giddiness and had vomited once. He had then become drowsy, refused food and been very constipated. He had been in hospital ten days previously for examination of the renal tract. At this time the right kidney had been found enlarged with a kink in the right ureter. On admission the patient had been very drowsy. He had lain on his back with his eyes half closed, had been able to answer questions quite intelligently, but had at once lapsed into semiconsciousness. The pupils had been equal and had reacted to light and accommodation. No abnormality had been detected in other cranial nerves. The deep reflexes had been present and active and the plantar reflexes flexor in type. No rigidity had been present in the neck and Kernig's sign had not been elicited. The temperature had been normal, the pulse rate 70 and the respiratory rate 18 in the minute. Dr. Fairfax had seen

the patient for the first time on March 4, 1927. He had then been semi-comatose, but had answered questions intelligently when roused, he had been very irritable and had resented any attempt at examination. Definite rigidity of the neck had then been present. Very little urine was being passed and occasional incontinence had been noticed. Lumbar puncture had been performed and 18 cubic centimetres of fluid, containing six lymphocytes per cubic centimetre, had been removed. The cerebro-spinal fluid had not reacted to the Wassermann test. On March 6, 1927, Kernig's sign had been present. On March 11 the patient's mental condition had not changed. He had been quiet during the day, but restless at night. He had refused food and had been fed by the nasal tube. The temperature had risen to 37·8° C. (100° F.). On March 13 incontinence of both urine and fæces had been noticed. On March 16 the mental condition had improved, the patient had kept his eyes opened and had complained that he saw double on looking to the right. Movements of the eyes had appeared normal. After this improvement had gradually occurred and had been maintained with the exception of one attack of drowsiness. He had gained control of the bladder and rectum. On April 18, 1927, after being up in a chair for four days, he had walked a short distance. At this time his intelligence had appeared to be normal, his speech had been slow. There had also been noted a definite Parkinsonian expression with nodding movement of the head, coarse tremor of tongue and hands, some rigidity of arms and legs and slowness of movements. When the patient walked, the legs had been moved in a stiff manner. The deep reflexes had been present and active and the plantar response flexor on both sides. The patient had been discharged from hospital.

Four weeks after discharge the patient had been readmitted. His wife had stated that after a fortnight, during which his mental condition appeared to be clear and during which he was getting stronger, he had begun to complain of continuous headache, had become dull mentally and had taken no interest in his surroundings. For the week prior to admission he had taken very little food and he had not been out of bed. He had had incontinence of urine and fæces for three days. Three days prior to admission *herpes zoster* had appeared as a rash in the third left dorsal area. Examination of the patient on admission had revealed that he lay on his back with his eyes half open and with an expressionless face, that he would put out his tongue and close his eyes, but would not answer questions. No abnormality had been detected in the cranial nerves, but a coarse tremor of the tongue had been present. General weakness of the limbs with some rigidity had been noted, more in the right arm and leg than in the left. A coarse tremor had been present in all the limbs. The deep reflexes had been present and active and the plantar response flexor in type. Neither rigidity of the neck nor Kernig's sign had been present. On lumbar puncture thirty-five cubic centimetres of clear fluid had been withdrawn. This had contained ten lymphocytes per cubic centimetre, 0·08% of glucose and chlorides 0·71%. The albumin and globulin in the cerebro-spinal fluid had been increased.

Dr. Fairfax said that he regarded the two patients as having suffered from *encephalitis lethargica*. In the first patient unusual mental changes had been present and in the second mental degeneration had occurred rather earlier than usual.

### Porro's Cœlio-Hysterectomy.

On behalf of Dr. H. H. Schlink, DR. R. ANGEL MONEY showed a female patient, aged thirty-one years, who had been sent into hospital on May 20, 1927, with the diagnosis of a six months' ectopic gestation which had ulcerated through the abdominal wall and was expelling amniotic fluid.

Dr. Schlink had not seen the patient until she was under the anæsthetic and the following history had been supplied by the admitting officer. The patient had been operated upon about fifteen months previously for a ruptured ectopic gestation and about two months later a lump had appeared on the lower part of the abdomen and "the stomach" had appeared to drop. The lump had gradually increased in size and two weeks prior to admission she had noticed an ulcer about 5 centimetres (two inches) below the umbilicus, and for the last few days watery fluid had been discharging from it. She had had amenorrhœa for six months and had been experiencing movements in the abdomen and getting labour pains every quarter of an hour. She had six full-time children, the first being ten years of age and the last two years.

On examination Dr. Schlink had found a ventral hernia, involving the whole of the previous section wound, with a six months pregnant uterus in the sac. The pressure of the uterus had caused an ulceration of the hernial sac, thus creating a hole the size of a five-shilling piece through which the uterus could be seen and the abdomen could be explored.

The opening had been split the full length of the old scar and except for a few slight omental adhesions around the neck of the hernial sac. The peritoneum of the abdomen and sac had seemed quite healthy. It was strange that no peritonitis had been existent except for a slight amount of ascitic weeping which accounted for the watery discharge from the abdominal fistula. The fact that the patient took no precaution to keep it clean or to protect it from her clothing showed how resistant the peritoneum was to the ordinary saprophytic organisms of the body. Dr. Schlink had considered that the case furnished one of the few indications for Porro's cœlio-hysterectomy as the danger of sepsis following repair of the enormous hernial sac had been too great to justify an attempt at an ordinary Cæsarean section. He had therefore removed the pregnant uterus by a supravaginal hysterectomy and had done a radical repair of the hernia. The specimen was displayed.

As Dr. Schlink had suspected, the wound had suppurated, but had not given way. The patient was convalescent. Had the child been removed and the uterus left behind, there would have been a very grave danger of puerperal sepsis as the edge of the ulcer had looked unhealthy and had displayed some pus formation and a placental site would not be so resistant to organisms to the same degree as the peritoneum.

### Obstetrical Films.

Dr. Money also exhibited on behalf of Dr. Schlink a series of obstetrical films in which the action of the musculature of the uterus was diagramatically represented.

### Tertiary Syphilis.

DR. C. G. MCDONALD showed four patients, who illustrated the varied manifestations of tertiary syphilis.

The first was a thin man, sixty-one years of age and an engine-driver by occupation, who suffered from syphilitic periostitis and gastric ulcer. He had been admitted on May 17, 1927, complaining of pain in the epigastrium during the previous nine weeks. The pain had radiated towards both scapular regions and had been relieved by alkalis. Practically each day he had vomited, but there had been no hæmoptysis and no melæna. His appetite had been poor and during the nine weeks he had lost 2·7 kilograms (six pounds) in weight. His bowels were constipated. He had had a painless swelling over the middle of the anterior aspect of the left tibia for a period of eighteen years. He had suffered from cough with expectoration for at least ten years. He had contracted syphilis forty years before.

Physical examination had revealed no definite abnormality in the alimentary system. He had suffered from *pyorrhœa alveolaris*, but there had been no tenderness, no rigidity and no palpable mass in the epigastrium. The spleen had not been palpable and the liver had been within the normal limits of dulness. There had been no abnormality in the nervous system. The pupils had been small and equal, but the reactions to light and accommodation had been brisk. The knee jerks had been active and equal. The other systems had been free of abnormal physical signs. Neither albumin nor sugar had been present in the urine.

A fractional test meal administered prior to admission, on May 11, 1927, had revealed an almost normal curve with a high resting free acidity, the specimen being thick, foul-

smelling and full of coffee-ground material. Blood had been present in considerable quantities. No lactic acid had been present.

An opaque meal had been administered on May 19, 1927, and Dr. Sear had reported the presence of a very large chronic ulcer crater on the middle third of the lesser curvature of the stomach, apparently fixed in position; the duodenum had been normal. There had been an "evacuation residue" at the end of eight hours. The report on the skiagraphic appearance of the swelling of the left tibia had been that it suggested the presence of osteitis of the tibia with probable gummatous formation. Although no clinical findings had been obtained on examination of the heart or lungs, X ray examination had revealed considerable dilatation of the ascending portion of the aortic arch, such as was frequently found in syphilitic aortitis and there was much mottling of the bases of both lungs which apparently represented the shadows of much fibrous overgrowth in the lung tissue, possibly of syphilitic origin. The blood serum had yielded a strong reaction to the Wassermann test.

The patient had been treated with a diet consisting of milk and milk foods and an alkaline powder administered before meals. Intramuscular injections of two cubic centimetres of "Muthanol" had been given at intervals of four days. After five injections the patient's epigastric pain had ceased, he had begun to enjoy a more liberal diet containing fish, vegetables, bread and a little meat and a further opaque meal had then shown such an extraordinary shrinking of the ulcer that it was difficult to demonstrate the position of the former ulcer crater. The swelling on the anterior border of the tibia had become almost level with the bone surface.

Dr. McDonald pointed out that the existence of tertiary syphilis in a patient suffering from gastric ulcer was no proof that the ulcer was itself luetic in origin, but the evidence in that case in favour of the diagnosis of syphilitic ulceration was unusually strong and justified his opinion that the ulcer was a further manifestation of the presence of spirochætes in the patient's body. Syphilitic ulcer of the stomach was usually associated with diminished or absent acidity, but in the case mentioned the acidity was practically normal. However, the remarkable change in the radiographic appearance of the ulcer and the disappearance of the patient's symptoms after antisyphilitic treatment for a period of a few weeks were sufficient justification for the diagnosis. The response to treatment was, after all, the main guide as to whether the ulcer was of the usual peptic nature or whether it was syphilitic in origin.

Dr. McDonald's second patient was a man, aged thirty-six years, suffering from tertiary syphilis and advanced osteoarthritis. He had been admitted to the Royal Prince Alfred Hospital on three previous occasions, namely in 1918, in 1921 and in 1926. On his admission on March 7, 1918, he had complained of pain and weakness in the left hip, right arm and right shoulder, right hip and left shoulder, gradually advancing over a period of four years. The lumbar and thoracic regions of the spine had been found to be immovable in a flexed position; the cervical spine had also been rigid; there had been no movement at either shoulder joint or either hip joint; but movement of the knee joint had been normal. The patient had suffered for about ten years from asthma. Examination of the sinuses and urine had failed to reveal a focus of infection. The blood had reacted strongly to the Wassermann test. He had improved after four intravenous injections of "Salvarsan." He had been discharged, but had been readmitted on March 7, 1921, suffering from bronchopneumonia. The joint lesions had not advanced to any considerable extent. In 1923 an operation had been performed on the patient at the Lidcombe State Hospital, the appendix being removed. After the operation he had suffered from another bout of bronchopneumonia. On June 10, 1926, he had been admitted to the Royal Prince Alfred Hospital for the third time, complaining of pain in the umbilical region of the abdomen which was continuous, but marked by severe exacerbations. He had not been constipated nor had he vomited. Examination *per rectum* had revealed an indefinite mass high up in the rectum. An opaque enema, administered shortly afterwards, had revealed a narrowing and irregularity of the descending colon, suggesting the presence of a neoplasm.

Examination of the patient at the time of demonstration after his admission for the fourth time, showed him to be thin and of small stature. His back was stiff and greatly bent; his neck was rigid and the head was diverted to the right, the appearance at first suggesting the presence of torticollis. He had a curious sidling gait; the movement at the ankles and the knees was excellent, but ankylosis at the hip joints prevented his walking in a natural fashion. As a result he propelled himself forward by shuffling his feet forwards and inwards. The cervical, thoracic and lumbar regions of the spine were completely ankylosed with considerable accentuation of the thoracic curve. There was slight movement at the occipito-atloid and atlo-epistrophoid joints. Both shoulder joints were fully ankylosed; both hips were ankylosed in flexion and adduction, but the elbow, wrist, knee and ankle joints on both sides were freely movable. Examination of the respiratory system revealed the presence of a "small-chest emphysema," the amount of expansion being not more than 2·5 centimetres. The breath sounds were everywhere vesicular, but many rhonchi were audible over the whole chest. The apex beat of the heart was in the fifth left intercostal space ten centimetres (four inches) from the middle line and systolic murmurs were audible at the aortic and mitral areas. The systolic blood pressure was 120 millimetres of mercury, the diastolic 60 millimetres. The alimentary system manifested no abnormality beyond a hernia in the appendicectomy wound. His pupils reacted to light and accommodation and his knee jerks were active. The blood had again reacted strongly to the Wassermann test. Six intravenous injections of "Novarsenobillon" had been administered, after which the blood had failed to yield a Wassermann reaction.

Dr. McDonald did not claim that there was any relationship between the osteoarthritis and the syphilitic infection, but he insisted that the extensive damage done by the former disease was related to the low resistance of the patient who had suffered for many years from unrecognized syphilis.

The third case was one of extensive aneurysm of the aorta in a man of forty-seven years, born in Sweden, who had been a ship's carpenter for many years, but for the last fourteen years had followed his occupation on land. He was a man of big stature. He complained of attacks of pain over the præcordium for about two years, increasing in severity during the last three months. He had little or no dyspnœa. He denied venereal infection. The apex beat was present in the sixth left intercostal space, 16·25 centimetres (six and a half inches) from the middle line. There was a definite pulsation in the second right intercostal space; a tracheal tug was present. Aortic and diastolic murmurs were audible over the aortic area. The radial pulsations were unequal in volume, the right radial pulse being much stronger than the left and exhibiting the typical Corrigan character. The systolic blood pressure in the right radial pulse was 170 millimetres of mercury, the diastolic 75 millimetres; the systolic blood pressure in the left radial pulse was 115 millimetres, the diastolic 85 millimetres. Capillary pulsation was pronounced in the finger nails and in the mucous membrane of the mouth. The pupils were equal and reacted to light and accommodation. The knee jerks were present and equal. Clinically the patient was suffering from aneurysm of the ascending and transverse portions of the aortic arch and a skiagram revealed a fusiform aneurysm involving these portions. The blood had reacted strongly to the Wassermann test.

The last patient shown by Dr. McDonald was a man of thirty-six years, suffering from *tabes dorsalis* and syphilitic osteitis of the lumbar spine. He had been previously exhibited by Dr. Sinclair Gillies at the corresponding meeting of the previous year (see THE MEDICAL JOURNAL OF AUSTRALIA, July 10, 1926).

The chief interest of that patient lay in the unusual association of *tabes dorsalis* with a tertiary lesion involving the spine. No distinction, however, could be drawn between the joint conditions described by Charcot involving

the large joints of the limbs and a lesion such as the one demonstrated involving the spinal portion of the osteoarthritic system.

### Abscess of the Lung.

DR. ALLAN S. WALKER showed a female patient, aged thirty-four years, who had been ill for about nine years. She was suffering from a bronchiectatic cavity in the lower lobe of the right lung, rather near the mediastinum, its origin being attacks of measles and influenza. The patient had had several attacks of hæmoptysis, at least one being of dangerous extent and for some time past she had been coughing up at least two hundred and forty cubic centimetres (eight ounces) of sputum *per diem*. When the patient was first seen by Dr. Walker eighteen months previously, her general condition had been very bad. Recently, however, she had improved greatly in health, though her symptoms had not been relieved. Tubercle bacilli had not been found in the sputum. As well as the right basal cavitation revealed both by X ray and clinical examination, there had been a certain amount of general bronchitis in the left lung, the peribronchial mottling in the skiagram being increased. In order to ascertain if there were any cavitation in the left lung, X ray examination after "Lipiodol" injection had been used. Twenty cubic centimetres of "Lipiodol" had been injected just below the cricoid cartilage. Dr. Walker demonstrated the skiagrams. He pointed out that the cavitation in the right lung could be clearly seen. In addition the bronchi in the left lung were well outlined and, although there was some slight dilatation, there was no evident cavitation.

Dr. Walker said that the patient had displayed one of the less usual complications of this procedure. She had become rather distressed and cyanotic, but the symptoms had soon disappeared. He claimed that the case demonstrated the advantages of examining the lungs by this method. Surgical measures had been adopted. Mr. Poate had performed pneumolysis on the right lower lobe and it was hoped that the compression of the cavity would ameliorate the patient's condition.

### Hypertrophy of the Bladder and Dilatation of Both Ureters.

DR. J. I. C. COSH showed a pathological specimen consisting of the bladder and ureters from a child two months old who had been ill for three days. He pointed out that the bladder and ureters had undergone considerable hypertrophy. He expressed the opinion that the condition was the result of phimosis. On the patient's admission to hospital a diagnosis of chronic intussusception had been made, but further examination had revealed that the mass palpated in the abdominal cavity consisted of the kidneys and ureters.

DR. A. H. TEBBUTT discussed the specimen and drew attention to the fact that the dilated ureter was the size of a thumb.

DR. J. C. STOREY differed from Dr. Cosh in regard to the causation of the condition. He thought that the condition resulted from infection of a congenital hydronephrosis. He pointed out that the child had been able to pass urine and that at *post mortem* examination it had been possible to pass a probe along the urethra without difficulty. At the autopsy a student had drawn attention to a fold of mucous membrane in the bladder which was situated near the urethral orifice. It was difficult to say whether this fold of mucous membrane had been concerned in the causation.

### A New Cephalotome.

MR. H. R. G. POATE demonstrated Souttar's cephalotome, a new instrument which he had just received. This instrument was specially designed for cutting an osteoplastic flap.

### Skiagrams.

DR. H. R. SEAR showed an interesting series of skiagrams of the skull. The conditions protrayed included pneumocranium, thickening of the skull in a patient whose serum reacted to the Wassermann test, gumma of the skull, osteomyelitis of the skull, *leontiasis ossea*. Dr. Sear pointed out that *leontiasis ossea* was generally held to be a rare disease. This was not the case as far as Australia was concerned. He had had experience of fourteen cases in his own practice.

DR. P. PARKINSON also showed a series of skiagrams of lesion in the gastro-intestinal tract. He discussed the condition of polyposis of the stomach.

## Obituary.

### PETER ANTHONY RICHARD LALOR

DR. PETER ANTHONY RICHARD LALOR whose death occurred at Sunbury, Victoria, was born in 1888. He was the son of the late Dr. Joseph Lalor and grandson of the late Peter Lalor who led the "Eureka Stockade" and afterwards became the Speaker of the Legislative Assembly of Victoria. He was born at Richmond, Victoria, and was educated at Xavier College, Kew, and at the Melbourne University.

After completing his medical course, he became a resident medical officer at Saint Vincent's Hospital, Melbourne, and then was appointed the first medical officer at the Duntroon Military College, under the late General Bridges.

He left there in 1916 and after a short time in general practice joined the Victorian Lunacy Department as junior medical officer at Ararat. He served in many of the hospitals for the insane of that State and was Medical Superintendent of the New Norfolk Hospital for the Insane, Tasmania. He returned to the Victorian Lunacy Department at the end of the war. At the time of his death he was Senior Medical Officer at Sunbury.

He took a great interest in all forms of insanity, particularly in epilepsy and published papers on that subject and on the treatment of puerperal insanity in THE MEDICAL JOURNAL OF AUSTRALIA. He was an excellent clinician and devoted his whole time to the improvement of the condition of his patients.

Among his colleagues he was beloved by all who knew him. He had the true Irishman's wit and knowledge of human nature. He made many friends and had no enemies. For twelve months prior to his death his friends were anxious about his health and his end came rapidly on June 28 from heart failure at the early age of thirty-nine years. He leaves a widow and four children.

His death will be regretted by everyone with whom he came in contact, and the Department loses the services of a valuable officer.

## Correspondence.

### THE TREATMENT OF OSTEOMYELITIS.

SIR: I shall be much obliged if you will kindly place this letter in your journal.

The report on my remarks on osteomyelitis at the British Medical Association meeting at Warragul on June 18 instant read as if I treated chronic and acute osteomyelitis in the same way. The object of the operation for the two conditions is entirely different, for the chronic condition to obtain if possible primary union, for the acute to obtain drainage and release pressure. Even in the acute stage I think it advisable to loosely stitch periosteum and wound, as long as stitches do not interfere with complete drainage.

Thanking you in anticipation.

Yours, etc.,
HORACE PERN, M.R.C.S., L.R.C.P.

Leongatha, Victoria,
August 7, 1927.

## THE COLLEGE OF SURGEONS OF AUSTRALASIA.

THE Honorary Secretary and Treasurer of the College of Surgeons of Australasia wishes to announce that the list of Foundation Fellows of the College is now complete. The Council is prepared to receive nominations for fellowship. Nominations must be made by three Fellows of the College on a form to be obtained from the Honorary Secretary, 13, Collins Street, Melbourne. All surgeons and surgeon specialists interested are referred to the records of the foundation of the College of Surgeons of Australasia which appeared in THE MEDICAL JOURNAL OF AUSTRALIA, January 22, 1927, page 134, and March 5, 1927, page 349. Copies of the By-Laws of the College, lists of the Foundation Fellows and other information may be obtained from the Honorary Secretary.

## Books Received.

MODERN MEDICINE, ITS THEORY AND PRACTICE, edited by Sir William Osler, Bart, M.D., F.R.S., Re-edited by Thomas McCrae, M.D., Assisted by Elmer H. Funk, M.D.; Volume IV: Diseases of the Respiratory System, Diseases of the Circulatory System; 1927. Philadelphia: Lea and Febiger. Sydney: Angus and Robertson, Limited. Royal 8vo., pp. 1011, with illustrations. Price: $9.00 net.

OVERCOMING TUBERCULOSIS; AN ALMANAC OF RECOVERY, by Gerald B. Webb, M.D., and Charles T. Ryder, M.D.; Third Edition, Revised; 1927. New York: Paul B. Hoeber, Incorporated. Demy 8vo., pp. 94, with charts. Price: $2.00 net.

## Diary for the Month.

AUG. 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
AUG. 23.—Illawarra Suburbs Medical Association, New South Wales.
AUG. 24.—Victorian Branch, B.M.A.: Council.
AUG. 25.—New South Wales Branch, B.M.A.: Branch.
AUG. 25.—South Australian Branch, B.M.A.: Branch.
AUG. 26.—Queensland Branch, B.M.A.: Council.
SEPT. 1.—Section of Orthopædics, New South Wales Branch, B.M.A.
SEPT. 2.—Queensland Branch, B.M.A.: Branch.
SEPT. 6.—Tasmanian Branch, B.M.A.: Council.
SEPT. 7.—Victorian Branch, B.M.A.: Branch.
SEPT. 7.—South Sydney Medical Association, New South Wales.

## Medical Appointments.

Dr. Ernset Kirsner (B.M.A.) has been appointed Public Vaccinator at Benalla, Victoria.

* * * *

Dr. James A. D. Nish (B.M.A.) has been appointed Public Vaccinator at Moonee Ponds and Ascot Vale, Victoria.

* * * *

Dr. A. P. Davis (B.M.A.) has been appointed Quarantine Officer at Port Hedland, Western Australia.

* * * *

Dr. George William Parramore (B.M.A.) has been appointed Government Medical Officer at Holbrook, New South Wales.

## Medical Appointments Vacant, etc.

FOR announcements of medical appointments vacant, assistants, *locum tenentes* sought, etc., see "Advertiser," page xx.

HOME AND TERRITORIES DEPARTMENT, MELBOURNE: Medical Officer, Public Service, New Guinea.

MANLY COTTAGE HOSPITAL, NEW SOUTH WALES: Resident Medical Officer, Assistant Honorary Medical Officers (Two).

## Medical Appointments: Important Notice.

MEDICAL practitioners are requested not to apply for any appointment referred to in the following table, without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

| BRANCH. | APPOINTMENTS. |
|---|---|
| NEW SOUTH WALES: Honorary Secretary, 30 - 34, Elizabeth Street, Sydney. | Australian Natives' Association.<br>Ashfield and District Friendly Societies Dispensary.<br>Balmain United Friendly Societies Dispensary.<br>Friendly Society Lodges at Casino.<br>Leichhardt and Petersham Dispensary.<br>Manchester United Oddfellows' Medical Institute, Elizabeth Street, Sydney.<br>Marrickville United Friendly Societies Dispensary.<br>North Sydney United Friendly Societies.<br>People's Prudential Benefit Society.<br>Phœnix Mutual Provident Society. |
| VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne. | All Institutes or Medical Dispensaries.<br>Australian Prudential Association Proprietary, Limited.<br>Mutual National Provident Club.<br>National Provident Association.<br>Hospital or other appointments outside Victoria. |
| QUEENSLAND: Honorary Secretary, B.M.A. Building, Adelaide Street, Brisbane. | Members accepting appointments as medical officers of country hospitals in Queensland are advised to submit a copy of their agreement to the Council before signing.<br>Brisbane United Friendly Society Institute.<br>Stannary Hills Hospital. |
| SOUTH AUSTRALIAN: Secretary, 207, North Terrace, Adelaide. | All Contract Practice Appointments in South Australia.<br>Booleroo Centre Medical Club. |
| WESTERN AUSTRALIAN: Honorary Secretary, 65, Saint George's Terrace, Perth. | All Contract Practice Appointments in Western Australia. |
| NEW ZEALAND (WELLINGTON DIVISION): Honorary Secretary, Wellington. | Friendly Society Lodges, Wellington, New Zealand. |

Medical practitioners are requested not to apply for appointments to positions at the Hobart General Hospital, Tasmania, without first having communicated with the Editor of THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales.

## Editorial Notices.

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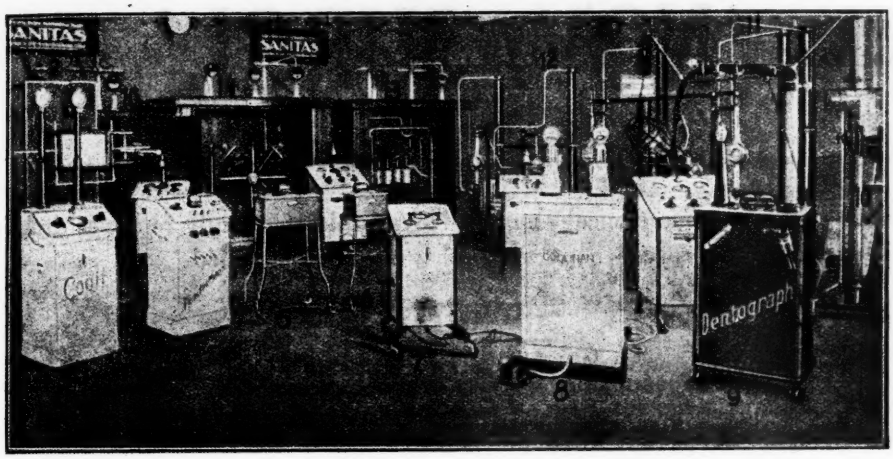


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*Recent Clinical References—Lancet*, Nov. 14th, 1925. *Prescriber*, Dec., 1925.
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